CLINICAL HEART DISEASE

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Fourth Edition, Illustrated

W B SAUNDERS COMPANY
PHILADELPHIA AND LONDON
1951

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W B SAUNDERS COMPANY

PHILADELPHIA

Preface to the Fourth Edition

Progress in our knowledge of heart disease has been rapid and valuable During the past five years much of both physiologic and therapeutic importance has been discovered There have been great advances in surgery of the heart, especially in the field of congenital heart disease Even the problem of acquired valvular disease is again engaging the attention of the surgeons, a problem that was attacked some twenty-five years ago but which had been forsaken The discovery of penicillin and other antibiotics has revolutionized the treatment of bacterial endocarditis and has transformed a previously fatal form of heart disease to one that is generally curable The use of anticoagulants in the treatment of coronary thrombosis has presented the medical profession with a tool that appears to decrease the immediate mortality of this condition Although this method of treatment is cumbersome and may possibly need further evaluation, it is sufficiently promising to deserve our careful attention

Apart from the great practical therapeutic advances that have taken place, our knowledge has been enhanced by means of the new diagnostic tool, i e , catheterization of the heart This has aided greatly in the diagnosis of various types of heart disease, especially in deciphering the different forms of congenital heart disease Further study of unipolar and precordial leads in electrocardiography has also made diagnosis of coronary artery disease more precise These various additions to our knowledge have been incorporated in the present edition They have necessitated much revision of the previous text, including a completely new discussion of the subject of electrocardiography For the latter I am greatly indebted to Dr Harold D Levine who has taken charge of that department at the Peter Bent Brigham Hospital and who has drawn freely from the written and spoken word of Wilson, Ashman and others

I am also grateful to Dr Lewis Dexter for suggestions concerning matters pertaining to congenital heart disease and catheterization studies, a field of work that he is prosecuting vigorously, and to Dr C B Favour for his advice on the subject of bacterial infections of the heart The painstaking task of constructing the index has been performed by Dr Bernard Lown to whom I express sincere appreciation Finally I wish to thank the publishers, W B Saunders Company, for their patience and cooperation

SAMUEL A LEVINE

Boston Massachusetts
January, 1951

Preface to the First Edition

The purpose of this book is to present in a simple form the important aspects of the diagnosis, prognosis and treatment of heart disease It is meant to appeal to the general practitioner, and in so far as the information or the points of view that it contains are applicable at the bedside and available to any intelligent physician, just so far will it be useful No attempt has been made to cover in detail the entire field of cardiovascular disease Larger textbooks have appeared recently that have done this adequately Nor does it contain any bibliographic references For the most part opinions have been adopted that are shared by present day authorities on the subject When apparently unorthodox views are presented, I alone must bear the blame for error, if time proves these views to be incorrect Where questions of opinion or speculations are involved, I have tried to draw the distinction between fact and surmise This should not detract but rather add to the interest of a medical treatise, for unproved impressions often precede by years established dogma

It cannot be said that the arrangement of the chapters follows any usual plan Each chapter may be regarded as distinct in itself and as a brief treatise on that subject The advantage of this is that they can be read independently In fact, many of the chapters represent the essence of individual papers that I have published in the past twenty years with the help of various men working at the Peter Bent Brigham Hospital and reflect, therefore, the results of personal intensive study of the problems involved

After the introductory chapter the various important types of heart disease are considered When specific or peculiar modes of treatment arise they are taken up as they come along, reserving the general subject of the treatment of congestive heart failure for the end Special topics that concern the practitioner, which merit emphasis, are discussed separately For example, because systolic murmurs are present in many forms of functional and organic heart disease and in fact even in normal individuals a special chapter is devoted to their clinical significance Similarly, acute cardio vascular emergencies arise under a variety of circumstances with and without organic heart disease and so, rather than discuss them in each chapter dealing with the respective type of heart disease, a special one is given over to this topic In this way the reader can review all the types of cardiovascular emergencies for which a physician may be hurriedly called Although the chapter on Clinical Electrocardiography was inserted at the very end, it may prove more useful to many to read it first

Some repetition has seemed necessary and advisable in order to spare

the reader from referring too frequently to one part of the book while reading another In discussing rheumatic heart disease, auricular fibrillation, for example, has to be considered It also is taken up as a complication of acute coronary thrombosis, hyperthyroidism and other conditions Nevertheless, when it is reviewed in the chapter on Clinical Electrocardiography, a brief summary is made of all the conditions in which auricular fibrillation is likely to occur Apart from avoiding this necessity of constant reference to different chapters, such a method has an added advantage It helps to give the reader two different modes of approach in medical diagnosis One may start from a given known finding, like a certain irregularity of the heart or clubbing of the fingers, and review what the various causes may be, or begin with a known disease such as coronary thrombosis and predict what kind of complications may arise Such repetitions, therefore, can only serve a useful purpose

The hope is that this volume will prove practical By this is meant that it will be easily understood and useful It may seem that certain parts receive more than their proper share of space and emphasis In general, points have been emphasized if they were simple, applicable at the bedside and of direct value to the patient Little time needs to be spent in a discussion of those subjects or phases of medicine that are already well understood A consultant with any extensive experience quickly finds out what is known and what is overlooked by the general practitioner From this experience he can readily sense the emphasis that is needed in teaching

If I may be permitted to digress a bit, I should like to express some views about our current methods of pedagogy in American Medical Schools Inasmuch as the main purpose of our schools is to train men to go out into the active practice of medicine we should keep constantly in mind that type of teaching that is practical and useful The minority of our students, who are to become teachers and investigators, must and do receive postgraduate training in their respective fields The initial undergraduate course, however, should be the same for all This curriculum seems to lack a proper distribution of time with insufficient attention to that type of teaching that is most useful For example, many hours are given to discussions concerning a subject like cancer of the pancreas which is entirely irremediable and too little to tumors of the spinal cord which are often completely curable The former, of course, is more common, but the latter are more important because they are amenable to effective treatment Granted that a medical student cannot be taught all we know about medicine in four or six years, it is more important, when he goes out into practice, that he should not overlook a case of spinal cord tumor with paralysis that has been diagnosed as amyotrophic lateral sclerosis or multiple sclerosis than to recognize a malignant growth of the tail of the pancreas Likewise, it is much more important that a physician should be able to recognize the thyrocardiacs who are masked as heart patients and suffer invalidism (so readily preventable), than to be able to make an early diagnosis of subacute bacterial endocarditis Until more is known about chronic arthritis and chronic nephritis it might be well to spend less time in our

teaching of these subjects and more time with a rare condition like hyperparathyroidism, because the limb pains, renal insufficiency and other disabilities due to the latter can be readily eradicated or prevented by appropriate treatment In a word, the first purpose in teaching is that the practicing physician should acquire that information which is directly helpful in the care of the patient This does not mean that clinical investigations and laboratory research concerning the unsolved problems should be discontinued A certain part of our profession must be constantly engaged in such effort.

Another aspect of medical education pertains to the simplification of medical diagnosis In teaching hospitals and medical centers, elaborate laboratory facilities are readily available for diagnostic purposes After an extensive *constructive differential diagnosis* has been built up, one possibility after another is eliminated by various tests When that same house officer or student goes out into practice, he recalls the numerous possibilities involved in a given set of circumstances, but he no longer has the x ray to rule out tuberculosis, a Wassermann test to eliminate syphilis and a blood culture to dismiss the diagnosis of septicemia What simple clinical bedside methods remain to enable him to establish a temporary working diagnosis? In other words, how is he to disentangle the complicated differential diagnosis without putting the patient to great expense? This type of clinical teaching has been neglected, for there are simple methods that can be used in what might be called the *destructive differential diagnosis*, which the older or more experienced physicians have learned and which they are really practicing consciously or unconsciously A physician finds that a patient has a palpable spleen and fever Among the various conditions to be considered is subacute bacterial endocarditis He learned in his hospital training that a positive blood culture would establish the diagnosis, but that a negative one does not eliminate it He has not been taught, however, that if there are no murmurs whatever, he can with fair assurance dismiss the diagnosis of subacute bacterial endocarditis This finding he can obtain in one minute and with no expense to the patient This merely illustrates one example, of which there are many, where simple methods enable one to rule out possible diagnoses It would be desirable if our medical teachers paid more attention to this type of instruction

A further difficulty in our teaching concerns the completeness or thoroughness of the examination There are numberless tests and signs for various diseases The practitioner cannot perform them all every time he sees a new patient There is not enough time nor can the public afford the necessary expense Therefore we must not only teach these various procedures, but we should emphasize more than we do in our schools when these procedures should be carried out For example, a systolic thrill in the aortic area is an extremely important sign of aortic stenosis, and yet this sign is often overlooked It can be missed when it is slight, because then it has to be detected by a special technic, i e , placing the palm over the upper sternum with the patient upright and holding a deep expiration Physicians cannot and need not go through this procedure with all patients,

but should be urged to do so only if there is also a fairly loud basal systolic murmur Similarly, determining the visual fields is a specialized examination and will not be performed by most practitioners However, it can be emphasized that, if there is some reason to suspect a pituitary tumor, a simple test for bitemporal hemianopsia can be performed in one minute by any physician Moving a pencil on each side of the patient while he is looking forward and ascertaining when he begins to notice its movements will serve as a gross test of bitemporal hemianopsia A further example is coarctation of the aorta We must teach not only what the condition is, but under what circumstances it should be particularly sought If a routine x-ray examination were made of the chest in all adult cases this diagnosis would not be overlooked This is impracticable We can emphasize that it needs to be thought of in all those who have hypertension, particularly in younger individuals, and if pulsations of the abdominal aorta or femoral arteries are diminished or absent, then further search for the evidence for or against this diagnosis should be made, even including the x-ray In other words, teachers need to emphasize and simplify, more than has been done, those sets of circumstances in which special procedures either simple or complicated need to be carried out

I want to take this opportunity to express my lasting gratitude to Dr J H Pratt, who first excited in me an interest in heart disease while I was an undergraduate student I also wish to thank Dr A E Cohn of the Rockefeller Hospital for first teaching me the experimental method as it might be applied to the study of cardiac problems All this would not have been sufficient if my chief, Dr Henry A Christian, had not afforded me every opportunity during the subsequent years for developing these interests I well recall the early days in 1913 and 1914 when Dr Christian first set up the electrocardiograph in the Peter Bent Brigham Hospital, having no one to turn to when this part or the other would not function After giving me my earliest instruction concerning this new apparatus and the subject of electrocardiography, he set me off on my own From then on I have remained constantly in debt to him for his stimulus and guidance in my work

Much of the joy and stimulus has come from the undergraduate students, whose insatiable curiosity and perplexing questions must ever keep the teacher's interest alive, and from the many house officers and resident physicians of the Peter Bent Brigham Hospital who have helped me in these studies during the past fifteen years We little realize the constant acquisition in knowledge that we experience from the casual and more spirited conversations with our intimate colleagues and medical friends This is one of the most characteristic and laudable aspects of our great profession Among a host of such friends I cannot refrain from acknowledging my enduring gratitude to Dr Frank N Wilson of Ann Arbor, Dr Tinsley R Harrison of Nashville, Drs Paul D White and Soma Weiss of Boston, Dr R W Scott of Cleveland and Sir Thomas Lewis of London From all I have learned a great deal

I also wish to thank Dr F Van Nuys of Weston, Massachusetts and

Dr W D Stroud of Philadelphia for their help in reviewing the manuscript and giving me the benefit of their criticism

Considerable time and effort have been saved by the kindly services of Miss Bertha I Barker, who has done all the technical work in electrocardiography at the Peter Bent Brigham Hospital these past twenty years I also wish to acknowledge my obligations to the Oxford University Press for permitting me to use some of the figures in Chapter 21 that were previously published in their System of Medicine

SAMUEL A LEVINE

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I

Introductory Considerations

Considerable knowledge has been gained since the turn of the century concerning the normal and abnormal changes that occur in the heart and the peripheral part of the circulation. One may ask whether or not this knowledge has improved the methods of treatment in actual practice. This question is often put with the inference that therapy has remained at a standstill and that progress in our understanding, though interesting to physiologists and teachers and somewhat tedious to students, has merely made the subject matter clearer and has put diagnosis on a more positive basis. Even if the latter alone were true and the diagnosis of heart conditions were now more accurate, a distinct advance of a practical nature would have been made. However, as will be demonstrated, prognostication, although still difficult, has become much more definite and, in certain respects, treatment more efficacious. There are now patients suffering from certain heart affections who are treated effectively, for whom improvement may be expected and, in some instances, complete restoration of health obtained, whereas a generation ago similar conditions were entirely overlooked or improperly understood and utterly beyond the help of any therapy that was then available. It will become clear in subsequent chapters that even the ablest clinician of not so long ago was helpless before some of the problems that now respond dramatically to treatment.

These advances have resulted from the more careful clinical study of our patients, utilizing the common bedside methods of observation, the pathologic study of autopsy material and the newer data that have come to us from the laboratory. With respect to the latter type of knowledge, we owe a great deal to the pioneer investigators, such as Mackenzie, Wenckebach, Einthoven and Lewis, who established modern cardiography on a scientific basis as a result of the introduction of the polygraph and the electrocardiograph. The use of basal metabolism determinations in clinical work has also materially helped our therapy. These and other laboratory procedures have fortified our knowledge so that, as a result of their use, we are now successfully treating patients suffering from certain conditions that were previously regarded as hopeless. Reference is made at this point to the recent improvement of our knowledge and to the means by which the advance was obtained, to combat the view that seems to be prevailing in the minds of many that laboratory methods have come to occupy too

prominent a position in our medical study An understanding of both the purely clinical and the more intricate laboratory aspects of disease are absolutely necessary for a proper approach to the diagnosis, prognosis and treatment of heart disease, and the slighting of one method or undue emphasis of the other will diminish the accuracy and value of our work

Aims in the Treatment of Heart Disease

At the outset, the proper aims in the treatment of heart disease must be appreciated Unlike other conditions in medicine, most sufferers from heart disease cannot be cured The disease generally is a chronic one, and the purpose of intelligent care is the prolongation of life, the diminution of suffering and the increase of mental and physical efficiency of the patient If the difference between correct and incorrect advice given to a patient with early heart failure is a matter of two to five years of added life, then proper treatment renders much more aid than most of the unhappy sufferers of cancer obtain from the thousands of surgical operations that are performed for their relief In addition, there is an increasingly large group of heart patients who present problems in which knowledge of the proper treatment saves life and effects complete restoration of health whereas, with lack of that specific knowledge, fatalities occur To be sure, such instances are uncommon, but to the few who succumb it is little comfort that these conditions are rare and therefore we should be ready to render this invaluable service when the occasion arises

There is a further aim to strive for in accurate study of heart disease which result purely from correct diagnosis I have reference to distinguishing organic from functional heart disease Many patients have been considered as suffering from structural heart disease because of certain signs or symptoms that we now know are benign This results in great and unnecessary economic loss and unhappiness and frequently in the perpetuation, aggravation or actual production of a cardiac neurosis which might have been cured or prevented at the outset if the condition were understood In other words, many sufferers from functional heart disease owe their disability to the inaccurate diagnosis made by some physician and to the effect produced by the fear and worry which such diagnosis engenders

A final purpose that one hopes will be more important in the future than it is at present is the possible prevention or diminution in the incidence of heart disease that may follow a sound understanding of its problems To be sure, we are now constantly hearing the cry of prevention from the lay public and the medical profession It seems that with our limited available information, too much is being promised by our medical brethren with regard to the prevention of heart disease Although much is being said, little that is effective has as yet been accomplished, but the great importance of the subject warrants the tremendous agitation that is current

Normal Circulation

Before taking up the discussion of heart disease, it may be well to review briefly some of the simple events upon which a normal circulation depends

The main functions of the circulation are the distribution of oxygen and other nutrient and essential constituents through the capillaries to the tissues throughout the body, and the elimination of noxious products mainly through the lungs and kidneys Let us at this point trace the different steps in the flow of blood within the body The venous blood returning from the periphery enters the heart through the superior and inferior venae cavae into the right auricle After an appropriate interval of diastolic filling of the auricle, during most of which time the tricuspid valve between the right auricle and ventricle is open, the auricle contracts and slightly less than one fifth of a second later, the ventricle contracts It must be appreciated that most of the blood, in fact about seven-eighths of it, goes from auricles to ventricles during diastole before the auricles contract, merely because of differences in pressure in the two chambers and the effect of gravity Only a last bit of blood is ejected by auricular systole which gives the ventricle its final stretching before it contracts When the right ventricle contracts, the tricuspid valve closes and the pulmonary valve opens Blood is therefore sent through the pulmonary artery to the lung capillaries There the essential change is the liberation of carbon dioxide and the absorption of oxygen in the alveoli of the lung, i e , the venous blood becomes arterial The blood then returns by way of the pulmonary veins to the left auricle The same movement of blood is going on in the left side of the heart, that was described above as taking place in the right side, from left auricle through the mitral valve to the left ventricle, only in the one case the blood is venous and in the other it is arterial When the ventricles contract, the mitral valve closes the aortic valve opens, and the arterial blood leaves the left ventricle through the aorta to enter the systemic circulation and to nourish the various organs of the body The blood returns from the capillaries through the veins back to the heart, only to start the cycle all over again The flow of blood is essentially a dynamic phenomenon and results from differences of pressure in one part of the system as compared to another

The above events recur with a rhythmic regularity under normal conditions at about the rate of seventy times a minute The disturbances in this rhythm that occur in certain normal and abnormal states are taken up in Chapter 21, familiarity with which is essential to a precise understanding of the treatment of heart disease These events produce two heart sounds that may well be described as lub dub or the first and second heart sounds The first heart sound is essentially the result of the contraction of the ventricles and the closure of the mitral and tricuspid valves, and the second sound is the result of the closure of the semilunar valves (the aortic and pulmonary) It has been maintained by some investigators, notably Dock, that the first sound is entirely valvular and that the contracting muscle itself produces no sound The interval between the first and second heart sounds is systole and that between the second and first is diastole The length of the former is approximately two thirds of the latter It will be seen later that in some cases, in order to avoid overlooking important findings in the heart that are to be heard by auscultation, it will be necessary

for the physician to train himself to listen to one of the four features independently of the others With a little training it is not difficult to dissociate from one s mind everything except the quality of the first or second heart sound Likewise, certain murmurs that occur in systole or diastole will be heard only if the mind is concentrated for a given length of time on the interval between the first and second heart sounds or between the second and first sounds I have emphasized this particular point because it has frequently happened that important diagnoses were overlooked as a result of aimless and more casual auscultation instead of concentrating attention upon a single element at a time while listening to the heart

Forms of Heart Disease

Before taking up any discussion of heart disease, it would be well to classify the general paths along which a heart may be diseased and the varying ways by which such disease may become manifest A very common affection of the heart results from deformation of the valves Such abnormalities may produce a regurgitation of blood through valves at a time when they should be closed or a constriction of the valves impending the free flow of blood from one chamber to another when they should be wide open The point of view I should like to emphasize at this time is that damage to valves is of great importance in undermining the efficiency of the circulation apart from the health of the heart muscle To express this somewhat differently if we assume the valves to be seriously injured at a time when the heart muscle is normal, progressive heart disease and failure of the circulation may yet take place because of the mechanical embarrassment that exists

A second form of disorder develops from changes in the musculature of the heart entirely apart from the integrity of the valves Here, as a result of alterations in the coronary blood vessels or of more diffuse damage to the heart muscle from toxins or certain poisonous substances circulating in the body, heart failure may result Good examples of this are coronary thrombosis or diphtheritic heart disease Furthermore, the heart muscle may fail functionally even when not significantly diseased, if there is marked hypertrophy In this case the blood supply to the heart may be relatively insufficient for the thick muscle fibers An entirely different form of heart disease occurs when bacteria start growing on the valves of the heart Here the heart muscle may be normal and efficient and the mechanical embarrassment to the circulation as a result of the valvular deformations of trivial importance The victim is nevertheless suffering from an affection of the heart which is almost always extremely grave It must be clear, however, that in this condition the disease presents itself with the picture of infection and sepsis and not as circulatory failure, whereas in ordinary myocardial or valvular disease the patients are apt to complain of varying degrees of shortness of the breath or chest pain

Disease of the pericardium may embarrass the circulation either as the direct result of the inflammation that is present in acute pericarditis or by the mechanical impediment to the normal free movements of the heart that

follows a pericardial effusion or pericardial adhesions Much in the same way, extracardiac conditions such as thoracic tumors that produce pressure on the heart or great vessels or emphysema that produces increased pressure in the pulmonary circulation may affect the heart

Another form of heart disease, which is comparatively rare, is that which results from congenital abnormalities Here, as a rule, the musculature is essentially normal but the chambers of the heart, its valves or partitions, or the large blood vessels are improperly constructed The result is either an impediment to the flow of blood in the normal manner or an admixture of venous and arterial blood because of defects in the septa that divide the right and left sides of the heart

Apart from the foregoing structural changes that account for most forms of heart disease there are disturbances in the mechanism of the beat itself which present distinct problems in diagnosis and treatment Such disturbances occur in normal people as well as in patients who also have structural disease of the heart For example, a perfectly normal individual may suddenly develop a paroxysm of tachycardia under such circumstances that very disastrous results may ensue Here the mere acceleration in rate enfeebles the circulation although there is no disease of the heart muscle or valves and no infection A similar situation may develop in a patient who already has mitral stenosis Then the embarrassment of the circulation may be more serious and develop more quickly Such abnormalities in the mechanism of the heart may properly be regarded as functional and will be taken up in detail later

There is a final condition which goes by a variety of terms that will also deserve our consideration I have reference to functional heart disease and cardiac neurosis These conditions were very prevalent during the First World War and had one or another of the following names soldier s heart, effort syndrome, disorderly action of the heart (D A H), neurocirculatory asthenia (N C A), irritable heart, functional heart, or nervous heart

The disability of which the patient will complain in one form of myocardial disease may be quite different from that in another Dyspnea is generally the most prominent symptom of cardiac failure whether it occurs in a patient with valvular or myocardial disease, however, when there is localized coronary artery disease producing angina pectoris, there may be no dyspnea whatever and only chest pain Likewise, the heart may be intoxicated as a result of hyperthyroidism without any dyspnea Here the heart is actually hyperactive and the primary complaint may be palpitation The inference from all this is that there can be no single method of testing the health of the heart This explains why the various functional tests of the heart that have been devised many of which are still being extensively used, have proved so unsatisfactory Most of these tests utilize the effect of some effort on the heart rate, the blood pressure or on the production of dyspnea in order to determine the health of an organ like the heart, which has so many different ways of expressing its abnormalities It would be just as impossible to decide that the brain is normal by finding that hearing or vision is not disturbed As a further illustration of this difficulty,

a patient may have a history of serious syncopal attacks with complete heart block (Adams-Stokes disease) and yet when put to a test of effort will show no dyspnea or pain whatever The reason for this apparent anomaly is that the main disturbance in function in this particular instance is one of conduction of impulses, and the other functions of the heart are essentially normal Functional tests in current use are for the most part tests of physical fitness and not of cardiac disease Many individuals with normal hearts may manifest a poor response to effort and, contrariwise, those with definite well-compensated heart disease may show a normal or excellent response The differences are due to the degree of physical training or to variations of nervous stability in different individuals and do not measure the present or future status of the heart itself The above will suffice to throw some light on the present status of functional tests of the heart and to indicate that the proper appraisal of problems in heart disease necessitates a complete survey of all factors that may have any possible bearing on the situation

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Rheumatic Fever

The term ' acute rheumatic fever has long been used in clinical medicine, but is an extremely unsatisfactory one The disease is often subacute or chronic rather than acute There may be no rheumatism or pains, and fever may be very slight or absent The term rheumatic state ' has come into use of late and has the advantage that it focuses attention on a ' state or peculiarity of the host, and yet a case of smoldering chorea need have no rheumatism There is not even a constant pathologic finding, for the Aschoff nodule in the myocardium or elsewhere which is so characteristic is not always present This is one instance in which it might have been advantageous to have a disease bear the name of some famous physician until its exact etiology were discovered, a custom in medicine that is often perplexing to students and practitioners In this chapter rheumatic infection is meant not to include infectious arthritis, although in some cases the former seems closely allied to the latter

Rheumatic fever is the most important infection that is directly related in a causative sense to the development of heart disease, particularly in younger people With this is included chorea or St Vitus s dance The exact etiology of this disease is not known although the streptococcus is thought by many to be the cause The evidence, however, is very conflicting and the question is best regarded for the present as unanswered It is very likely that certain hemolytic streptococci play a role in this connection, as the disease so frequently follows in the wake of sore throat, tonsillitis or other streptococcic infections The occurrence of epidemics of rheumatic fever in camps or institutions, where large groups are crowded together, following a widespread hemolytic streptococcus infection of the throat affords strong evidence in favor of the view that there is some causal relationship between the streptococcus organism and rheumatic fever The exact relationship, however, is not well understood

Much has been written of late about the allergic nature of rheumatism comparing it to asthma, hay fever and urticaria, only that the sensitivity in the cases of the latter is to proteins and in the former to bacteria and their products, particularly streptococci This conception has aided in the understanding of some of the manifestations of the disease It can explain why joints may swell in response to a sore throat without the presence of

bacteria in the joints Under such circumstances certain tissues (e g , the skin or joints) may respond to infection or streptococci in distant parts of the body (e g , the tonsils, teeth, or sinuses) because of local alterations in sensitivity This point of view gives a different aspect to the idea of focus of infection" in its relation to rheumatism

At the outset it becomes very important to have a clear understanding of this particular infection and its various manifestations The rheumatic infection often appears seven to fourteen days after some primary illness or upset This primary cause generally is a streptococcus infection, like a sore throat, but may be an ordinary surgical operation the injection of some foreign protein, a chilling or exposure Scarlet fever is one of these trigger mechanisms that may start this series of events, but it cannot be regarded as a cause of rheumatic fever or rheumatic heart disease On careful analysis it will be found that in only 2 to 5 per cent of cases of scarlet fever are there cardiac complications This percentage corresponds to that of rheumatic individuals in the general population It will generally be true that the cardiac murmurs which follow scarlet fever occur in that small number of patients who showed mild arthralgic symptoms about ten days after the onset of the disease In other words, the scarlet fever infections uncover those individuals who are constitutionally rheumatic or vulnerable It is not unlikely that if the scarlatinal infection had occurred at some other time when the particular host was not vulnerable it would not have resulted in a rheumatic bout

We are all familiar with the typical attack of acute rheumatic fever in a child or a young adult who is suddenly afflicted with painful joints which are tender, warm, swollen and red, the symptoms jumping from one joint to another in rapid succession During such a condition there is a moderate fever and slight leukocytosis This may last a few days, a few weeks, or even months We are also familiar with the typical attack of chorea in which the child insidiously develops involuntary nervous muscular movements or twitchings of a peculiar character Both these conditions may properly be regarded as different manifestations of the same rheumatic infection, for they frequently occur together, they attack the same type of individual and in a sense produce the same disabilities in the heart When either of these two conditions occurs in a typical form, it is very easily recognized However, when the symptoms are slight, they are frequently overlooked both by the patient s family and by the physician, remain unrecognized, and lay the same foundation for the subsequent development of rheumatic heart disease I have often seen children who have appeared somewhat nervous or fidgety, in whom it was extremely difficult to tell from the symptoms that chorea existed, and yet in whom there was sufficient evidence of a subsidiary nature to make it certain that a true Sydenham s chorea was present The same has frequently been true of mild cases of rheumatic fever Here the patient may only have vague aches and pains in the limbs, often called growing pains,' and yet, because of these same secondary manifestations of the rheumatic infection, the true character of

the underlying disease became evident We must, therefore, be ready to make the diagnosis of rheumatic infection in many atypical cases

Atypical Cases

The failure to recognize atypical cases of rheumatic infection accounts for the fact that in many instances outspoken valvular disease of the heart is seen in adults when no past history of rheumatic fever or chorea can be uncovered In fact, if we take a condition like mitral stenosis, which I believe is due to only one disease, namely, rheumatic infection in only about 50 per cent of the cases will there be a definite history of rheumatic fever or chorea In the other 50 per cent, I believe that rheumatic infection occurred years previously, but presented itself in an atypical form, though with sufficiently characteristic features to be recognized if these unusual aspects of the rheumatic infection had been appreciated

We must look upon the rheumatic infection as a very protean disease, in many respects similar to syphilis Apart from the more commonly known organs that are involved, attention has been drawn to rheumatic lesions in the lungs, kidneys and blood vessels of other structures It affects almost the entire body A child has an attack of rheumatic fever at the age of 9, recovers satisfactorily, and then presents himself twenty years later with mitral stenosis A young adult has a chancre at the age of 20, and presents himself with aortic insufficiency twenty years later Chorea may be regarded as the nervous manifestation of rheumatism, as meningitis is of syphilis Both diseases have cutaneous symptoms, i e, the nodules and erythema multiforme of rheumatism as compared to the secondary rash or later tertiary syphilide In both, the heart is frequently involved The analogy may be carried further, for the joints and other organs are affected in both conditions It is also true that the predominance of one type of symptom or another varies considerably from patient to patient in both diseases In some syphilitics, the cutaneous features are very prominent, in others they are almost absent, and the central nervous system involvement is the outspoken lesion Likewise, in some rheumatics, the element of arthritis may be entirely absent and the nervous manifestation in the form of choreic movements may be the sole feature of the disease In others, there may be neither joint nor nervous symptoms, and the disease will be confined entirely to an affection of the heart or of the skin There are numerous instances in childhood in which the illness is characterized by gradual fatigue lassitude slight pallor, mild sweats loss of appetite and weight, and a slight fever without any limb pains or chorea Sometimes such a child will be considered as suffering from tuberculosis, possibly involving the hilus glands The rheumatic nature of the disease is often overlooked and in fact could be suspected only by the most careful consideration of the secondary factors of rheumatism In these cases it will frequently be found that the heart sounds are hyperactive or that a murmur over the precordium will be present, either of which should direct one s attention to the possibility of rheumatism One may be left in doubt as to the diag-

nosis only to see the child at some subsequent time go through a similar illness, this time associated with typical polyarthritis or chorea We must, therefore, not confine the diagnosis of rheumatic infection to those patients suffering from typical polyarticular rheumatism or St Vitus's dance

The various manifestations of rheumatism are best looked upon as differences in the type of response on the part of the host rather than differences in strains or virulence of the infection We all do not respond to the same insults in the same way This applies to psychic, bacterial or mechanical trauma One patient loses his entire fortune and then commits suicide, another after suffering a great financial loss takes to drink and a third grits his teeth and starts all over again Similarly one patient has a primary chancre and despite good treatment develops a stubborn skin syphilide A second with little or no treatment has few or no skin lesions and only becomes aware of his plight when twenty years later he has involvement of the central nervous system A third has no involvement of the nervous system but develops an aortic aneurysm Likewise two individuals cut their hands and blood flows One faints at the sight of the blood and the other has enough presence of mind to put his handkerchief over the wound These are differences in the response of the host and such differences are of extreme importance in rheumatic fever, for they explain the multiplicity of the symptomatology It also must be remembered that the host changes during different periods of life so that the response in childhood may be different from the response in adult life or old age

Detecting Atypical Forms

Appreciating the fact that the rheumatic infection need not appear in its typical form as polyarthritis or chorea, what means have we of detecting the atypical forms of this disease? There are numerous clues that have frequently been invaluable in diagnosis that may be called subsidiary or secondary features of the disease None of them is characteristic enough to be pathognomonic, for they are vague and may occur in many other diseases, but it is surprising how often, when considered in toto, they make up a distinct clinical picture In the first place I have reference to *epistaxis* It is well known that nosebleeds occur in many normal individuals and in a variety of diseases, but I know of no group of individuals who have repeated epistaxis as frequently as rheumatic children I do not refer to the nosebleed that comes from trauma The epistaxis here is spontaneous and may occur for some years before the outspoken attack of rheumatic fever takes place, it may occur during the active rheumatic infection while the child is sick in bed with fever and polyarthritis, or after recovery while feeling well and attending school Whether this is due to a peculiar vulnerability of the small blood vessels of the body in these individuals, or whether the actual cause of rheumatic fever which lurks in the body for many years produces specific pathologic changes in the mucous membrane of the nose, is not clear It must be appreciated, however, that the rheumatic infection has a predilection for synovial membranes It affects the endocardium, the pericardium, the pleura, the peritoneum, the synovial mem

branes of the joints, of the eye and possibly of the nose At any rate, a history of repeated nosebleeds, together with other features, of doubtful significance in themselves, should make one strongly suspect that the patient is rheumatic

Another symptom, although not quite as frequent as epistaxis, but one which may similarly be used in making a diagnosis of rheumatic infection is *repeated vomiting spells* The child may be ambulatory, attending school and suddenly have an attack of vomiting This is generally painless and accompanied by only slight nausea or none at all To be sure, vomiting is a frequent occurrence in many nonrheumatic children, but it has impressed me that recurrent attacks of vomiting are more common in this disease than can be accounted for as an accidental phenomenon Sometimes, with or without this vomiting, there is pain in the abdomen and tenderness One can readily see from this brief description how a child might erroneously be operated on for acute appendicitis because of the symptoms of nausea, vomiting, pain in the abdomen, slight tenderness, fever and leukocytosis I have personally seen several instances in which this mistaken operation had been performed It is in these unusual cases that one may be entirely dependent on other features of rheumatism to arrive at a proper diagnosis, such as hyperactive heart sounds murmurs, family history and nosebleeds

Familial Factor

Further peculiarities of rheumatism pertain to the family history and the constitutional type of the individual There is now no doubt whatever that there is a strong familial factor in rheumatism What is not clear is whether the high incidence of the rheumatic infection in members of the same family is due to a particular hereditary element or whether it is due to the fact that members of the same family are exposed to the same environmental influences If the disease has a contagious or infectious element in it (and well-established epidemics of rheumatic fever have been reported), it would not be surprising that two or more children in the same household should have the same disease Even if it is not contagious, but dependent upon factors like dampness unhygienic surroundings, overcrowding and diet, one would also expect to find an apparent familial instance of the disease I am inclined to the opinion that apart from the infectiousness of the disease there is a distinct hereditary predisposition In one family I know of two sisters and a brother who lived in different parts of New England, yet one or more of the children of each sibling has had rheumatic fever or chorea In this same family the children have had one or another form of rheumatic infection and their parents and grandparents have shown marked evidence of degenerative vascular disease, namely, hypertension and angina pectoris Here we have a striking example of familial vascular vulnerability, the children developing the infectious type of circulatory disease and the parents the degenerative form I have seen this combination of rheumatism in children and angina pectoris in the parents too frequently for the relationship to be entirely accidental The inference to b drawn at this point is, I believe, that there are families with vulnerabl

vascular systems and that this vulnerability is both to the infectious and the degenerative form of heart diseases

Apart from the familial predisposition to rheumatic infections there are rare instances of intra-uterine rheumatic involvement of the fetus It would be difficult otherwise to explain the finding of well-marked mitral stenosis in a child eight months old who showed this finding at autopsy This can be called an instance of fetal endocarditis

Constitutional Factor

Much attention has been given to constitutional or anthropologic features of the individual in relation to disease I have been interested in this problem in so far as it bears upon three distinct clinical conditions, namely, pernicious anemia, angina pectoris and rheumatism There seems to be no doubt now that there is a type of individual with fair skin blue, gray or light brown eyes and fair hair (prone to early grayness) that more readily develops pernicious anemia Highly pigmented individuals are only rarely affected by this disease In a similar manner, there seems to be a certain type that frequently develops angina pectoris I refer here to the well-set, stocky, strong man (mesomorph) who has always enjoyed good health In this type the muscles seem to be hard, the skin tight and the forearms well rounded rather than of flat configuration There are also some striking characteristics in the rheumatic cases It is surprising how many freckled and red haired children, frequently with hyperextensive fingers are seen in our heart clinics This is the type characterized in England as having the rheumatic diathesis Although I have not any statistical control studies, I feel quite certain that the incidence of such individuals among patients with heart disease exceeds that in the population at large Another finding of possibly less importance is the appearance of the sclerae Many of the rheumatic children show pinkish coloration of the sclerae, which is due to an increase in the number of capillaries that come in from the periphery toward the iris This peculiarity, however, may not be constitutional, but rather the result of infection The foregoing constitutional considerations, although not as yet established on a firm scientific basis, cannot be lightly brushed aside They seem to be important enough to deserve further study and have actually proved distinctly useful in diagnosis

Regional and Seasonal Distribution

Other peculiarities of rheumatism pertain to its regional and seasonal distribution There is no longer any doubt that rheumatism, meaning by this term rheumatic fever and its allied conditions, is distinctly more common in certain parts of the world than in others It is much more prevalent in New England, for example than in the southern states For some time this was not believed to be true and the apparent difference was explained by the fact that in any statistical study the terminology was confusing In the North the disease might be catalogued under the term 'rheumatic fever, and in the South under some other name such as infectious arthritis or polyarthritis When it was found that the incidence of mitral

stenosis at autopsy in a general hospital of a large southern city was about one tenth as great as in a similar hospital in Boston, one could not avoid the conclusion that rheumatism must vary a great deal in its frequency in different parts of the country No matter what the original infection was called in the two cities, if the disease were equally frequent, mitral stenosis, the common result of this disease, and a condition which is eventually fatal, would necessarily have been found with equal frequency in any large series of autopsies performed in the two places This regional difference in the incidence of the disease is important entirely apart from its possible bearing on the nature of the malady, for where the disease is prevalent, it makes it imperative for the physician to suspect its presence even on slight or doubtful evidence

The seasonal variations are also of some importance Until the specific etiology is known we shall have no explanation of many of the features and peculiarities of rheumatism Among these are the variations in the prevalence of the disease during the different months of the year The early spring is a particularly precarious time of the year, both for the development of new cases and for recurrent attacks in old cases There if a practical inference in this observation, for if we have any measures cs protection or prophylaxis that may be beneficial, it is during the months of February, March and April that these measures should be carried out most energetically

Cardiac Damage

Rheumatic disease is primarily important in so far as it affects the heart The acute problem, whether it be polyarthritis or chorea, of course produces a disabling condition, but one from which recovery is almost always complete It may last, however, a very long time The painful joints or the active chorea may come and go over a period of years This aspect of the disease is trying on the patient, his family and on the doctor, but they may all find comfort in the knowledge that eventually all this disappears The most distressing feature of the condition is the great frequency of cardiac damage, even when the original infection is apparently mild It is difficult to estimate how frequently the heart is involved, because one would have to define what is meant by heart damage If all the means that are now available to detect abnormality of the heart are utilized in studying this question it will appear that close to 100 per cent of the cases show some evidence of heart damage To be sure, many of these changes are slight and transient, others, although permanent, may produce no bodily discomfort to the patient nor diminish his future usefulness However, it is fair to say that more than half the cases subsequently suffer a more or less serious organic cardiac condition

It must be clear from the foregoing remarks that whereas the rheumatic infection may show manifold symptoms, it may also appear in a singularly pure form In one case there may be a great deal of polyarthritis, in a second there may be rheumatic nodules and vomiting and nosebleeds may occur, in another none of these symptoms will be present and only active chorea

will be noted The disease is the same in all instances, but the response of the body is different What is much more important is that in a fourth case none of these symptoms may be manifest, but there may be an acute affection of the heart Here again the cause is the same rheumatic infection

It is quite conceivable and logical to regard the rheumatic infection as a great deal more prevalent than has been recognized It can be compared to the situation that exists in relation to infantile paralysis In this disease there is reason to believe that for every case in which paralysis occurs, there are many others in which the same infection took place but no paralysis developed Likewise, may there not be numerous instances of rheumatic fever in which the rheumatism is either absent or very slight? There are probably frequent instances in which the child has a slight fever and sore throat, shows no appreciable arthritis, but develops a heart murmur Such patients would be considered as developing valvular disease of the heart not due to rheumatic fever, but rather as a result of a simple sore throat or without any known etiologic cause In other words, there are probably many mild infections, rheumatic in nature, which affect the heart, but which go unrecognized and later go to form that large group of patients with organic valvular disease in whom no past history of rheumatic fever can be obtained

Clinical Features

Let us now consider briefly some of the clinical features that the patient presents who comes down with a rheumatic attack The onset of the disease may be abrupt or insidious It often follows a sore throat or an acute tonsillitis The patient may, within a short time, complain of rather severe pains in one joint or another, show a moderate fever, and begin to perspire It is characteristic of this disease for one joint to clear up rather quickly, and for another to become troublesome In the fulminating case, the joints are extremely tender to the least motion, and there is swelling, redness and increased warmth of the affected parts Sometimes these symptoms recede spontaneously, or as the result of salicylate therapy In other cases, the arthritis remains refractory to treatment for a considerable period of time There are great variations in the severity, duration and stubborness of the joint manifestations Occasionally the entire illness seems to subside within a few days or a week, and the patient recovers More frequently there is an amelioration of symptoms, but the infection keeps smoldering for weeks, months, or even years During the acute stage of the disease, the patient is apt to develop some secondary anemia, and look pale In almost all cases, the heart is accelerated out of proportion to the degree of fever This is an important feature of the disease There is hardly any other condition in which the heart rate continues as rapid with a temperature of 100 to 101 F for as long a time as in rheumatic fever It is a common experience to see the heart rate continue around 120 for months with a fever of only one degree Even if practically all the symptoms have disappeared and the patient feels fairly well, a slight fever and a rapid heart may persist I have seen instances in which a temperature of about 100 F and a heart rate of

about 110 lasted for several years During all this time the patients were feeling quite well, attending school, and undertaking ordinary activities Finally, without any particular treatment, the slight fever and tachycardia gradually returned to normal There is no better proof of the chronicity of this disease than such experiences This also throws light on how the rheumatic infection may lurk within the body, smolder in a comparatively inactive way, and suddenly, without any evidence of a reinfection, flare up and new symptoms appear Reactivation is a term that well describes this peculiarity

At different times during the period of activity of the disease there is frequently pain in the precordium This sometimes is fairly severe and troublesome, and may occur without any clinical evidence of either pericarditis or pleuritis The heart sounds have a hyperactive quality This pounding of the heart troubles the patient, and he complains of palpitation The quality of the sounds resembles very much that heard in hyperthyroidism It is always a matter of great importance to determine, if possible, whether any part of the heart is being involved during the acute infection, and if so, to what extent Similarly one should try to foretell whether certain suspicious evidences of damage are likely to indicate a permanent structural injury or not In many cases, this is rather difficult, and at times impossible A more detailed discussion of this question will be taken up in the following chapter

Diagnosis

The typical case of acute polyarthritis with involvement of one joint after another and complete subsidence of the previously affected part is easily recognized The response of the fever and the pain to salicylate therapy is fairly characteristic but by no means pathognomonic In some cases salicylates are not very effective and at times other types of fever or pains respond to this drug In the atypical cases a complete knowledge of the subject will be necessary to identify the condition as rheumatic fever The family and past history, the season of the year, the occurrence of epistaxis or vomiting the feeling of listlessness and fatigue the presence of sweats or anemia the peculiar acceleration of the heart and the increased intensity of the sounds, the development of cardiac murmurs, the detection of a skin rash like erythema multiforme or erythema marginatum the presence of small rheumatic nodules in the scalp, elbows, feet and spine or over ligaments are all important aids in diagnosis The sedimentation rate of erythrocytes is increased but so it is in almost any infection and therefore is not particularly helpful It does aid in estimating whether or not the process once identified is still in the active stage, and with the presence of fever and leukocytosis may serve as a guide as to the length of bed rest Furthermore, there are peculiar immunologic reactions that occur with rheumatic fever During the week or two following the initial streptococcus infection those persons who are rheumatic or who are prone to develop symptoms of rheumatic fever are likely to show a high titer of antistreptolysin in the blood In fact, when such bodies do not appear, rheumatic

given in one dose by rectum in 50 to 100 cc of water If uncomfortable ringing of the ears develops, the drug may be omitted for a day or two and treatment reinstituted with smaller doses As the symptoms and the fever subside, the dose should be diminished It has been the custom on the part of many clinicians to keep the patient on a maintenance dose of sodium salicylate or aspirin after the acute stage of the disease has subsided This latter dose will vary with the size of the patient from about 0 3 gm to 1 or 2 gm a day

Coburn reported striking results following intravenous injections of very large doses of sodium salicylate, i e, 12 to 24 gm in 2000 to 3000 cc of solution, daily, for a few days He has devised a chemical method of measuring the concentration of salicylate in the blood The aim of this treatment is to obtain a level of 400 gamma per 100 cc of blood He has claimed that the joints improve promptly and the heart escapes injury More extensive experience with this method of treatment has failed to confirm the early enthusiastic reports It now is believed that the course of rheumatic fever is not significantly shorter on intravenous than on ordinary but adequate oral therapy Furthermore the possibility of salicylate poisoning must be considered

During the active stage of the disease, the painful joints should be protected by proper support in the form of pillows, and frequently it is necessary to place a hood around the feet and legs as even the weight of the bed covers may be distressing Bandaging the tender joints after applying oil of wintergreen may give added comfort Occasionally, the pains will be so distressing that sedatives such as codeine or even morphine may be advisable for short periods of time These details in the care of the patient are of considerable importance, as they are supportive and help to spare his vitality which is needed to combat an illness that frequently is chronic

There is no particular problem in the care of the bowels except that it is advisable that the patient have one movement a day, with or without the aid of any cathartic Inasmuch as rheumatic fever is frequently accompanied by a great deal of sweating, it may be necessary to change the patient s clothing frequently and to watch the condition of the skin The heart itself, in the vast majority of cases, needs no specific medication, although in patients who have precordial pain an ice bag may be helpful Although digitalis is often given because the heart is rapid I have seen no evidence of any beneficial effects except in rare instances when simultaneously with the acute infection there is congestive heart failure as well The matter of food is of considerable importance The diet should be liberal and nutritious One should welcome an actual gain in weight, for, in most cases, the patient is already undernourished I know of no reason for limiting the diet in any way Every attempt should be made to encourage extra nourishment The diet should be adequate in vitamins, especially in fruit juices, all the more so because it is thought by some that deficiency in vitamin C is an important factor in the development of rheumatic fever In fact, very recently it has been suggested by Massell that very large doses of ascorbic acid (1 to 4 gm daily) produce dramatic beneficial effects on active rheumatic fever

It is always a matter of considerable moment to decide how long to keep the patient in bed This, of course, will depend a good deal upon the severity of the illness and upon the extent to which the heart is involved One would prefer to continue bed care until there are no symptoms and the temperature, pulse, white count and sedimentation rate have been normal for at least one month In many cases, in order to accomplish this, the patient will have to be bedridden for many months, particularly if one waited for the normal heart rate to be resumed, for, as has been mentioned above, tachycardia may continue even for years When the disease persists and smolders, showing only slight evidence of activity, the practical problem becomes extremely difficult To obtain the desired result, one might have to confine the patient to bed for one or more years At some point in the course of the illness, the question of diminishing returns comes in In the average public clinic patient, if such a drastic procedure is carried out in the attempt to obtain a slight and somewhat questionable advantage by means of a time-consuming and troublesome plan of treatment, the child frequently loses years of schooling When recovery takes place, which might possibly have been accomplished without the loss of so much schooling the child finds himself handicapped economically in later years of life There is also to be considered the additional cost to the family of this prolonged medical care When the advantages from prolonged bed rest are obvious, the decision is simple Almost any sacrifice should be made if permanent and severe injury to the heart can be obviated It is only when this advantage is either very slight or doubtful that these economic features enter directly into the decision I often permit a child of humble means to continue at school after a previous trial of bed care, although there are slight fever and a few joint pains, whereas if the economic status of the family permitted private nursing and tutoring I should advise the child to remain in bed The importance of these considerations would be evident to any one who has charge of large numbers of clinic patients

Prophylaxis

This discussion would be incomplete without a word concerning prophylaxis The intimate relationship between sore throat and tonsillitis and the development of rheumatism has been known for many years Very frequently an acute attack of tonsillitis or sore throat initiates either the first attack of rheumatism or a recurrence This brings up the whole question of a focus of infection as the underlying cause in the persistence of this chronic disease The direct inference from the foregoing statements is that removal of tonsils would have a beneficial effect on the condition There has been a great deal of discussion *pro and con* concerning this matter At first it was hoped that tonsillectomy after the initial attack of rheumatism would actually diminish the number of recurrences that would take place and the degree of subsequent damage to the heart If this is true, it certainly is so only to a slight extent for we often see the disease persist unabated after the most careful tonsillectomy After this disappointing experience, it was thought that once the disease had started, removing the tonsils

was like closing the barn door after the horse had escaped It was then argued that the infection had spread from the tonsils and was lurking elsewhere in the body It would follow, therefore, if this premise were true, that by removing the tonsils in healthy children before the first infection took place, rheumatism would either be prevented or at least diminished in its incidence This, of course would require very elaborate and extensive statistical study Such a study has been carried out in Rochester, New York, where many thousands of normal children with tonsils and without tonsils have been followed over many years To date the results are only slightly indicative of beneficial effects so far as the diminution in the occurrence of rheumatism and rheumatic heart disease is concerned Certainly, there are large numbers of children who have their first attack years after tonsillectomy At present it would be fair to state however, that children who have had their tonsils removed are slightly less liable to have rheumatism in the future than those who have not had their tonsils removed

There are other practical aspects to the whole question of tonsillectomy that merit our consideration If this operation is to be done it is safer during childhood than in later years Although post-tonsillectomy lung abscess is a rare complication, it occurs much more commonly in adults than in children The same is true of subacute bacterial endocarditis which occasionally develops after tonsillectomy in adults with valvular disease, and almost never in children Finally, the operation is a much simpler procedure in children, requiring only a day or so of hospitalization These are additional reasons for early tonsillectomy in rheumatic children When a tonsillectomy of election is being contemplated it is better to have it performed in the month of May or June than in August, September or October, because during the latter months poliomyelitis is more prevalent and there is a possibility of developing a severe post-tonsillectomy bulbar type of poliomyelitis that might otherwise have been avoided

The discovery of sulfonamides, penicillin and allied chemicals has been an epoch making turning point in the treatment of many infections, including those due to streptococci It is obvious therefore, that they would be quickly used with the hope of treating rheumatic fever and to prevent rheumatic heart disease It was soon discovered that the sulfa compounds had no beneficial effect on rheumatic fever once the symptoms appeared, and even did not prevent their development if the drugs were given at the time of the previous sore throat This experience afforded further proof that there was a great deal more to the rheumatic state than the original streptococcus infection It then was thought that if the primary streptococcus infection could be prevented, the entire chain of events might be inhibited For this purpose a series of cases were given 0 5 gm of sulfadiazine three times daily as a prophylactic agent during the months of October through April Although this method of treatment requires watching for any untoward effects of sulfonamide medication, some beneficial results have been obtained If this treatment were to be given, it should be for the first year or two after the initial attack of rheumatic fever

A more promising but economically more costly procedu[illegible]

penicillin prophylactically One would have less fear of any toxic effects and there is reason to believe that the doses that can be used would be more effective One might employ 100,000 to 200,000 units three times daily during the most precarious months or throughout the year It is too early to know whether this method will prove worthwhile If it does it is hoped that the cost of penicillin will fall further so that it might become available to all who need it

A factor that interests me a great deal from the point of view of prophylaxis is the question of weight It seems to me that the obese child is rarely stricken with an initial attack of rheumatic infection This has been particularly true with regard to chorea, although many such children become obese after the disease has started if they progress favorably One may contend that the apparent relationship between the rheumatic infection and the lowered state of nutrition is that the latter is the result of the former rather than the cause of it It is difficult to offer scientific proof in this regard one way or another, and one would, therefore, have to depend for the present on general impressions However, as a result of observation of a great many rheumatic patients, I cannot avoid the conclusion that undernutrition is conducive to the development and the prolongation of this disease and that a diet particularly rich in proteins should be given to vulnerable children

This discussion brings up the relation between normal or average weights and optimum weights Doctors and parents often refer to standard weight tables when they consider the weight of children We must appreciate that the normal figures given in these tables are average ones, derived from thousands of so-called "normal" individuals These figures are, therefore, averages of good, bad and indifferent weights One should strive, not for average figures, but for optimum ones The best weight at any part of life may be quite different from the average I feel that the best weight for children and young adults is distinctly above the average, and as we shall see later in discussing degenerative heart disease, the best weight for the second half of life is distinctly below the average If this view is true, the practical inference follows that a deliberate attempt to keep infants and children overweight might actually diminish the ravages of rheumatic fever Such attempts might be more particularly applicable when other features like a positive family history or peculiar constitutional characteristics indicate that the child is more than ordinarily susceptible to rheumatism The same efforts in the dietary care should be carried out even after the first attack has occurred Recurrences, I feel, are less apt to develop, or are less apt to be damaging, if those who once had rheumatism gain weight or actually become somewhat obese

There are other measures of a preventive nature that deserve some attention It is a frequent experience that an attack of rheumatism quickly follows exposure to wetness and chilliness I have seen instances in which the first attack came immediately after the patient was soaked through to the skin in the rain or was chilled from lying down on cold, damp ground This should make us caution our patients to avoid getting their feet wet or their

body chilled, and we should call these matters to the attention of the parents of these children so that they may take appropriate prophylactic measures It is a simple matter for the physician to tell the patient or his parents that he ought to avoid sore throats or catching cold This is easier said than done No doubt upper respiratory infections have an intimate bearing on or are often responsible for initial or subsequent attacks of rheumatism, but we have, as far as I know, no certain or specific measures for avoiding these infections, except the general ones that pertain to bodily hygiene Mouth cleanliness and appropriate dental care should be carried out Much is being said about the importance of the teeth in their relation to *systemic disease, especially rheumatism and rheumatic heart disease* I cannot convince myself that I have ever seen an instance in which this causative relation existed as far as rheumatic heart disease is concerned Teeth have a more important bearing on the development of subacute bacterial endocarditis than they have on rheumatic fever Notwithstanding this doubt and skepticism as to the role of infected teeth, I do urge all these patients to keep their teeth in a satisfactory condition

Two other procedures, the value of which has not yet been demonstrated but which deserve attention, are vaccination against colds and x-ray treatment of the throat The former in the opinion of some authorities, will diminish the incidence of upper respiratory infections, and if this can be accomplished vaccination should prove of value The latter has some theoretical justification for its use We know that radiation can destroy adenoid tissue in the throat It has been used as a substitute for tonsillectomy by some physicians There are numerous miliary tonsils on the posterior pharyngeal wall that cannot be enucleated surgically, each one of which might be regarded as a possible focus of infection I have, therefore, advised radiation of the throat in some cases in which sore throats have persisted after tonsillectomy What good has been accomplished thereby in these cases is still uncertain There is a further use of the x ray that deserves consideration In a small number of cases a series of six daily spray x ray treatments given to the entire body has been of help for troublesome aches and pains of smoldering rheumatic fever The dose used is 25 r The beneficial results in such cases probably come from an effect upon the immune or reactive state of the host rather than from any antibacterial action of the x ray

An upper respiratory infection with fever deserves more than usual consideration in a rheumatic individual During an ordinary cold many of us continue our daily work with the hope that it will pass by without any further ado the risk is so great in an individual with a possible rheumatic infection that it is expedient that the individual should remain in bed until the illness is well over

A final consideration in the matter of prevention concerns the question of climate As has been mentioned earlier in this chapter, there are parts of the United States in which rheumatism is comparatively rare The *question arises whether we can utilize this factor in a prophylactic way* Will an individual who might come down with rheumatic fever while living

in Boston, for example, avoid the disease if he spends his life in Florida, Southern California or in a city like New Orleans, where the disease is less common? There are no statistical studies to give the answer to this question It is logical to think that the disease might be avoided in this way, but this has reference to patients who have as yet not suffered from the first attack The matter is quite different when we consider what might happen to a patient who has already had a rheumatic attack and then goes to a region where the disease is less prevalent In other words, will recurrences be less frequent in one place than in another, or does the fact that the disease has once obtained a foothold make it independent of external surroundings? I have on occasion advised parents to move their families out of New England to warmer climates when one of the children persisted in having recurrences of rheumatism despite all therapeutic measures that we carried out Although such instances have been too few in my experience to draw any definite conclusions therefrom, it seemed that good was accomplished when a change to a warmer climate was made However, uprooting the whole household is extremely costly and drastic, and naturally can be undertaken only by a few families It would be highly desirable if we could have more data in this regard, for, although costly, the move would be worthwhile, if effective The whole question of treatment and prevention of rheumatism and its complications presents a most difficult and unsatisfactory situation The solution, therefore, will have to await the discovery of the actual cause or the true nature of the disease

From a sociologic and public health point of view slow progress is being made and much more is to be hoped for There is already statistical evidence that rheumatic fever and rheumatic heart disease are on the decline in the United States Rheumatic fever is mainly a disease of urban populations and its incidence is directly related to factors such as crowding, dampness and poverty As these conditions are improved rheumatic fever will be more satisfactorily controlled In this regard there is reason to suspect that improved ventilation will help matters considerably as droplet infection appears to play some role in the spread of streptococcal infection

Prognosis

The ultimate prognosis of any single attack of rheumatism is most variable Recurrences are numerous and, in fact, are the rule In only a small number of instances does death occur with the acute stage of rheumatism and then it is as a result of a pancarditis, frequently associated with nephritis In half the cases, at least, permanent heart damage results It has been thought that the more frequent the attacks, the more likely there is to be a cardiac complication This may need some qualification for in many cases there are repeated bouts without any further cardiac involvement Some patients, in fact, suffer no permanent heart damage after several attacks Thus, in some instances, we might be led to believe that the "all or none" law applies, i e, if the heart is to be attacked the damage will be done in the first attack and the degree of cardiac involvement will be as great in those who have one as in those having multiple attacks The younger the

individual at the time of the first attack, the more likely the heart will be involved There are certain peculiarities in the outcome, depending on the original type of rheumatic infection, which at present remain unexplained Although we rightly regard chorea and rheumatic fever as merely different manifestations of the same disease, yet there are qualitative differences in the type of heart damage that results from them

When a child has chorea and no other manifestations of rheumatic infection (pure chorea) the heart is much more rarely involved than when rheumatic fever occurs with or without chorea This applies to the events during the immediate few years after the infection I do not believe, however, that chorea will prove to be as benign as it seems, if these same patients are seen years later I feel that many will still show valvular disease, in some because recurrent rheumatic infection will have taken place and in others even though there has been no subsequent infection In other words, the child who had chorea at the age of 11 may show no evidence of heart disease at the age of 15 and yet appear at the age of 30 or 40 with mitral stenosis, never having suffered from any relevant illness in the meantime I have not infrequently seen women over 50 with mitral stenosis who only then began to have symptoms of heart disease, who had had chorea in childhood and no subsequent illness It is baffling to conceive of what was taking place in the mitral valve all those forty or fifty years between the original illness and the development of significant valvular disease If chorea were as benign a disease as an early follow-up study would lead one to believe there ought to be a considerable number of adults who give a past history of chorea in childhood but who show no evidence of heart disease This has not been my experience

A further peculiarity of chorea is the predilection for involvement of the mitral valve when there is a cardiac complication Although the mitral valve is the most common one to be affected no matter what type of rheumatic infection may have been the cause, it is particularly so when the antecedent illness is chorea, for aortic involvement and pericarditis are rarely seen in those who have only had chorea

These qualitative differences in the cardiac complications are rather difficult to explain if we accept the prevailing view that rheumatic fever and chorea are due to the same underlying cause for one would then expect similar types of complications as far as the heart is concerned There are two possible explanations that may have a bearing on this In the first place, there may be different strains of organisms that cause rheumatism, one having a predilection for the nervous system and the other for the joints The organism with a predilection for the nervous system may more easily affect the mitral valve than the pericardium or the aortic valve One may draw an analogy between this state of affairs and syphilis It used to be thought that there were strains of spirochetes that had a predilection for the central nervous system and others for the cutaneous system The other possible explanation is that the virus is the same, but that the hosts differ There are certain other factors that lend emphasis to this latter view Chorea, for example, is more prevalent in the female than in

whereas the reverse is true with regard to rheumatic fever Here one might say that sex alters the type of response to the same virus, the nervous system of the female being more vulnerable than that of the male May there not be certain anatomic differences in the valves of the heart between those which develop mitral stenosis and those that do not or that develop aortic disease? Recent work has shown that in some individuals the heart valves contain blood vessels and in others they are absent, whereas some years ago heart valves were thought to be entirely devoid of blood vessels May it not be true that whether mitral stenosis develops will depend upon the presence or the extent of the blood vessels in the mitral valve, and that *those which are entirely or comparatively avascular escape?* Such anatomic considerations may account for the discrepancies in the kind of cardiac complications that follow rheumatic infections

CHOREA

The general considerations as to the treatment of rheumatic fever apply with equal force to chorea Just as the presenting complaint in the one, i e, painful limbs, may vary in degree from practically no pain whatever to extreme, exquisite pain, likewise the nervousness may be manifest in all gradations from merely a fidgety child to the extreme chorea insaniens Occasionally there is marked weakness and paresis of an affected limb I once saw a patient in whom the mistaken diagnosis of poliomyelitis was made During active chorea there is apt to be no fever or only a very slight rise in temperature and the heart rate is generally slow or at least not so accelerated as during an active rheumatism The treatment for active chorea is a prolonged period of rest in bed and general nursing care Although various medicines are used and each physician may have his preference, there is little evidence of a specific value that can be attached to any of them Some prefer bromides or barbiturates, others use arsenic in the *form of Fowler's solution, while a third group believe that sodium cacodylate* intramuscularly is of value At times the nervousness seems to be helped by daily warm tub baths As I have watched the various methods used I have felt that isolation in a hospital with good nursing, bed care and forced feeding were the main factors in recovery When the chorea is of the extreme or maniacal type morphia may be indicated or even avertin by rectum (70 mg per kg) may be given Fever therapy has been advocated and found beneficial in shortening the duration of active chorea The temperature of the body is raised to about 105 to 106° F by means of a hot box and kept at that level for several hours Such treatments are repeated once or twice at intervals of three to five days In all but rare instances recovery takes place, for the cardiac complications during active chorea are hardly ever of any great immediate concern The nervous twitchings may continued in a mild form with irregular exacerbations for several years

There is one type of chorea that deserves special mention, i e, the chorea of pregnancy Both rheumatic fever and chorea occur most frequently in the second decade of life, particularly during the ages of 10 to 13 When

once either form of the disease has appeared there is a marked tendency for subsequent attacks In some patients the same manifestations return year after year while others who first had chorea will later develop rheumatism or vice versa After the period of full growth has been passed and the patients have reached the age of about 18 years recurrences become much less frequent From then on, more purely cardiac complications become manifest with repeated bouts of fever Rheumatic pains and even outspoken rheumatism, however, are not at all rare in the third and later decades Occasionally even the first attack of rheumatic fever may take place during these latter decades Curiously this is hardly ever true of chorea I have not seen a single instance of chorea occurring for the first time in a patient past the second decade, except in the rare instances when it is associated with pregnancy Even then there probably will be found a history of some manifestation of previous rheumatic infection The great prevalence of chorea (and, for that matter, rheumatic fever) during the period of active growth of the child and the fact that chorea is never seen again except in association with pregnancy, brings to one s mind the possibility that the endocrine balance or the calcium metabolism is in some way related to the susceptibility to rheumatic infection During both of these periods there is a great disturbance of the glands of internal secretion and an unusual demand on the calcium metabolism in the production of the long bones of the individual during growth and in the laying down of new bones in the fetus during pregnancy

A final sidelight on the nature of the rheumatic infections is the peculiar seasonal incidence mentioned above The early spring seems to be the most prevalent time of the year both for first attacks and for recrudescences of the disease This has interested me a great deal and particularly so in the light of work on animals at the Rockefeller Institute that showed seasonal variations in the susceptibility to experimental syphilis It was found in this research that the weight of the various parenchymatous organs, particularly the glands of internal secretion, varied a great deal during different months of the year February and March were found to be months during which animals were particularly susceptible to experimental syphilis During these months moreover, the relative weight of certain glands was especially low It was also found that the calcium content of the blood varied in different months May not similar factors be playing important roles in human susceptibility to rheumatic infections? In this connection the following experience is of interest I once saw a young boy 11 years old, who had just come down for the first time with an attack of chorea It began during the month of February I learned from the boy s mother that her brother (the patient s uncle) had had chorea when he was 11 years old and that he was first taken ill with it during the month of February Thus, the same disease occurred in two members of the same family at the same age and during the same month It makes one think that perhaps there is some inherent hereditary defect in metabolism associated with growth or with the glands of internal secretion that renders some individuals particularly susceptible to some noxious agent that is fairly gener-

ally prevalent This conception that changes in the internal environment, especially the endocrine system of the host, may have a bearing on whether or not an individual will develop a rheumatic infection deserves more investigation than it has received, for it may suggest methods of prevention that so far have been wanting despite an enormous amount of bacteriologic and immunologic research It at least gives added meaning to some vague phenomena, such as "growing pains' and 'spring fever," that characterize this mysterious disease

It is not unlikely that much valuable information will come to light in the near future concerning the relation of the endocrine glands to many other diseases Excision of the testes has already been found to have a favorable influence on cancer of the prostate The fact that lupus erythematosus disseminatus is so overwhelmingly confined to women during their years of menstruation leads one to think that the ovaries play some role in this disease The disappearance of a stubborn acne of the face directly after radiation of the ovaries is another example The very low mortality of patients with untreated lobar pneumonia at the age of 10 in contrast to the high mortality of those at the age of 70 may be related to differences in the endocrine system Patients with thyrotoxicosis appear to have a much greater incidence of concomitant rheumatic heart disease and hypertension than other persons in the same environment Women have very much less arteriosclerosis than do men and live several years longer on the average Does this depend upon some function of the endocrine system? One might cite other illustrations, all of which lend support to the view that the endocrine glands may have an important influence on vulnerability to infections as well as to the development of some noninfectious or metabolic diseases

The above discussion concerning the possible relation between the endocrine system and rheumatic fever and other diseases is similar to one taken up in the first edition of this book in 1935 It is very interesting to see that recent developments have borne out this speculative prediction Since the discovery of the dramatic effects of Compound E on rheumatoid arthritis by Hench and Kendall this endocrine product has been administered for many other conditions with profound results It is much too early to draw any final conclusions, but already favorable improvement has been observed in acute rheumatic fever, lupus erythematosus and gout apart from the original observations on rheumatoid arthritis Because of the great scarcity of Compound E (a product of adrenal activity), adrenocorticotropic hormone (ACTH) has been used in a small number of cases and has produced somewhat similar results A critically ill child with acute rheumatic pericarditis improved dramatically in a few days following 10–20 mg of ACTH subcutaneously every six hours Similar beneficial effects were obtained in a case of lupus erythematosus which ordinarily has been refractory to all known forms of treatment The implications from this discovery are great and remain to be unraveled by future study It is obvious that Compound E or ACTH, which stimulates the adrenal to produce Compound E, are not antibactericidal agents In some way they must alter inflammatory tissue

response It seems that these endocrine products change the internal environment of the host so that more normal metabolic activity is restored, at least temporarily There is reason to hope that fundamental advances in our knowledge of many metabolic processes and new approaches in the treatment of hitherto incurable conditions will result from this work

3

The Development of Rheumatic Heart Disease Mitral Valve Disease

ACUTE RHEUMATIC CARDITIS

The development of rheumatic heart disease may best be studied by considering two aspects of the disease first, what is taking place during the acute rheumatic infection and, second, the progressive changes that occur later, which go to make up the whole picture of chronic rheumatic valvular disease During the acute rheumatic infection it is frequently difficult to tell whether or not the heart is being affected at all and whether certain changes that are noted indicate a transitory or a permanent cardiac damage Often one has to leave the question open, and delay a final decision for some months or years During this time the condition may possibly be called ' potential heart disease ' There are certain criteria, as we shall see, which afford quite conclusive evidence that the heart is involved There are other changes that are common accompaniments of many febrile reactions, and, therefore, have not the same significance The distinction between these two types of changes is a most important one to appreciate in following a case of rheumatism

While the active infection is going on there is naturally some fever and acceleration in the heart rate The tachycardia is frequently out of proportion to the degree of fever This is quite typical of a rheumatic infection, but need not indicate that the heart is seriously damaged or will show structural changes after the infection is over If the patient develops signs of circulatory insufficiency such as marked dyspnea, congestion of the lungs and liver, or peripheral edema which are not common during the acute stage of the disease, we must assume that some serious injury is taking place in the heart Even under these circumstances it is surprising how much improvement may eventually occur and how little may remain, after a long convalescence, to indicate any permanent organic heart lesion When these symptoms are severe and a satisfactory recovery occurs, we cannot avoid the conclusion that there must have been an acute rheumatic myocarditis, in which healing took place without impairment of cardiac function

Systolic Heart Murmur

The most difficult feature to appraise properly during the acute infection is the development of a systolic heart murmur We all are familiar with the fact that a very slight systolic murmur may be present during fever, whatever its origin, especially when the heart is rapid and hyperactive A more detailed discussion of the significance of a systolic murmur is taken up in Chapter 17 However, the louder the systolic murmur that one hears during acute rheumatic fever, the more likely it is to be permanent and to indicate some organic lesion This is more particularly true if a murmur of moderate intensity is present while the heart action is comparatively slow As the heart slows, what might be regarded as a benign or functional murmur ought to diminish in intensity, for with the slowing the hyperdynamic element becomes less prominent Loud systolic murmurs rarely disappear Faint systolic murmurs may disappear, but if they persist they may be practically disregarded The murmur of moderate intensity needs to be carefully followed and if it persists as the infection disappears, I believe that some injury, possibly minor in nature, has occurred although it need not incapacitate the patient in any way It is clear from this discussion that we must pay some attention to the loudness of the murmur and to its persistence When it is interpreted as indicating a structural damage, it generally points to involvement of the mitral valve, especially if its intensity is loudest in the apex region of the heart This may even be true when the point of maximum intensity is at the base, although this is not so certain We now know that the rheumatic infection may affect the wall of the aorta and this may add to the confusion of interpreting the causation of the basal systolic murmur

Disturbances in Conduction

A more certain indication that the heart is being affected during the acute disease is the finding of disturbances in conduction in the mechanism of the heart beat Not infrequently during ordinary bedside examination one detects an actual heart block, an occasional omission of an entire heart cycle or a dropping of the beat This indicates an acute myocarditis, for the conduction apparatus of the heart lies within the musculature, and there must be some toxic process or some structural damage, like an Aschoff nodule, affecting the auriculoventricular node or the bundle of His Although it has recently been shown that this delayed conduction may disappear on full doses of atropine indicating that it is vagal in origin, it is difficult to believe that such observations mean that the heart muscle has not been involved Minor evidence of this disturbance in conduction which does not produce an actual blocking of beats, may be detected by the use of the polygraph or the electrocardiograph Here it is found that the time it takes an impulse to go from auricle to ventricle is merely delayed, although the beats all reach the ventricle The normal conduction time for impulses to go from auricles to ventricles is less than one fifth of a second It is considered delayed if this upper limit is exceeded The heart will then

be perfectly regular and it might be impossible by bedside examination to discover this disorder Occasionally one may suspect it on detecting a gallop rhythm on auscultation, or on observing that the first heart sound is weak or fainter than it was formerly If careful studies are made of the heart during the acute infection, changes in conduction are found to be quite common, and they are one of the best proofs that the heart muscle is being involved, at least temporarily Fortunately, recovery from such damage is very likely to be complete since, when the infection has passed, the conduction disturbances tend to disappear Even in the rare instances, when some permanent defect remains as evidenced by the continued presence of a delay in the conduction time (P-R interval), the efficiency of the circulation may be perfectly normal

Electrocardiographic Changes

There are other changes of a qualitative nature, as shown by the electrocardiogram made during the acute process, that are fairly characteristic and that also indicate that the heart is being affected by the disease The changes to be sought for are evidence of auriculoventricular heart block or abnormalities in the configuration of or lengthening of the QRST complex This means of diagnosis is a more specialized one and can only be utilized by those clinicians who are carrying on electrocardiographic work Suffice it for the present to bear this possibility in mind, for occasionally such changes in the form of the ventricular complex (see Chap 21, Fig 171) may be helpful from a diagnostic point of view, and may aid in determining whether or not a certain vague infection is rheumatic in origin In fact, there will be instances in which the electrocardiographic changes will be the only evidence that the illness is of rheumatic origin A girl of 13 who complained of listlessness, nausea and vomiting was seen by her physician He found a slight fever (100 F), slight tenderness in the right lower quadrant and a leukocytosis of 14,000 The child was sent to the surgical service to be operated on for acute appendicitis A few hours after admission the temperature was only 99 F , and the white blood count was 12,000 The surgeon noted a faint abnormal sound over the precordium There was very little evidence of any abnormal condition in the abdomen, but the electrocardiogram showed a P-R interval of 0 30 second It seemed to me that the child had an atypical form of rheumatic fever of the abdominal type with a mild acute myocarditis, without any rheumatic pains On rest in bed and salicylate therapy the patient recovered and the P-R interval returned to normal (see Chap 21, Fig 111) When the abdominal pain and tenderness are more marked than in this case, an unnecessary appendectomy is likely to be performed

Enlargement of the Heart

In the attempt to determine whether or not the heart has become affected during an acute rheumatic infection, the question of hypertrophy, or dilatation, of the heart is a matter of considerable importance Although during the acute stage, it cannot be said that a heart that is normal in size is not

diseased, it may be safely accepted that if the size of the heart increases or is greater than normal, it is diseased In fact, we may make the generalization that enlargement of the heart is always a definite sign of disease of that organ There are important exceptions to this general rule Appreciable dilatation without hypertrophy occurs with severe anemia, especially in children This will naturally increase the percussion outlines and the x ray silhouette of the heart Such dilatation of the heart disappears completely when the blood returns to normal Dilatation without increase in weight of the heart is also found occasionally in acute toxic states, particularly when the heart muscle is affected It is, therefore, important to try to estimate the presence or absence of hypertrophy or dilatation Ordinarily this is done by percussion and palpation When it is possible to feel the apex impulse quite distinctly beyond the nipple line it is fair to assume that the heart is enlarged Frequently one is in doubt as a result of bedside examination whether the heart is enlarged or not, and further information in this regard may be obtained with the aid of the x-ray and the electrocardiogram The electrocardiograms may give indirect evidence of preponderant hypertrophy of one ventricle over another and occasionally may throw light on whether the auricles are dilated or hypertrophied Precordial electrocardiography is often more helpful in detecting hypertrophy of one or the other ventricle than the x-ray This type of data needs to be weighed most carefully, for there are numerous difficulties in interpretation If it is decided by one means or another that the heart has become enlarged during the acute process, we must conclude that the heart has been affected

Other Evidences of Acute Rheumatic Carditis

While following any particular patient during an acute rheumatic attack, if enlargement of the heart has not been noted if a systolic murmur is either absent or only slight and there are no obvious evidences of circulatory insufficiency, what other features are to be watched for? The development of a *pericardial friction rub*, which may appear any time during the acute illness, will, of course, be definite evidence of cardiac involvement Not only will the detection of a typical to and fro pericardial friction sound be proof of an acute pericarditis, but it may be assumed that in most such cases the heart muscle is also affected Another condition that can develop during the acute process of the disease is an *aortic diastolic murmur* heard in the aortic area and better still in the third left interspace near the sternum This aortic diastole murmur may be entirely absent on one day, appear as a faint blow a few days later and then be quite definite within a week or so When it appears it persists and is definitely indicative of an aortic regurgitation for it only rarely disappears A diastolic murmur of mitral origin is not to be expected to develop at this time, if we assume that the patient is now suffering from the first attack of rheumatism It takes months or years for contraction and constriction of a valve to occur, for this is a chronic scar tissue process, whereas an insufficiency or regurgitation of a valve may take place within a few days To produce the latter it is only necessary for the valves to become slightly retracted or distorted

by inflammatory reaction resulting in a slight incompetency That same incompetent valve years later may contract as a result of scar tissue formation and then be stenosed The important point is that in the acute infection we may look for insufficiency of valves, but if evidence of stenosis is detected, it is likely that the valve was previously injured and that we are not witnessing the first rheumatic infection but a recurrence

Occasionally in children, during acute rheumatic carditis, a faint, short *diastolic murmur* may be heard at the apex resembling the murmur heard in mitral stenosis Such murmurs must be interpreted cautiously for they may be due to dilatation of the heart or to some other mechanism and not to mitral stenosis, and they may disappear entirely They are not apt to have the same rumbling quality that is found in older patients with mitral stenosis In fact, errors of this type in the interpretation of the apical diastolic murmur as a sign of mitral stenosis are very rare in adults although not uncommon in children

The main pathologic evidence of acute or active rheumatic carditis is the finding of Aschoff nodules in the myocardium Such lesions are common in fatal cases of rheumatic fever in children However, they are by no means rare in patients dying of valvular disease over 40 years of age They are occasionally found in older cardiac patients without valvular disease, and even in patients who gave no clinical evidence of rheumatic fever or heart disability and died of some noncardiac cause

MITRAL INSUFFICIENCY

Let us assume that the patient did not develop any of the definite signs of heart involvement during the acute stage of the illness and that if there were minor changes, such as a delayed conduction time or a slight change in the form of electrocardiogram, they were transitory We assume that the patient had been in bed for some months with a smoldering fever, a rapid heart rate and vague fleeting pains in the limbs, and finally all these symptoms subsided Let us also assume that he was left with a systolic murmur of moderate intensity heard best at the apex and that when he recovered he was symptom free In the course of time he returned to his ordinary activities either at school or at work and felt quite well As we observe this patient during the following years, let us trace what possible changes may take place He may remain well, never have a return of rheumatic fever and always show a systolic murmur on examination Such a patient would either be denied life insurance in his later years, or be considered an increased risk and receive a high rating in his insurance examination Occasionally, the systolic murmur may gradually diminish in intensity Rarely, it may disappear entirely He may, therefore, live out his life as a normal individual and never be embarrassed by his heart This, I think, is the exception, unless with recovery no murmurs whatever remained

In a fair number of patients in whom all the findings on examination, including the systolic murmur, were considered benign, some years later, usually a decade or two, subacute bacterial endocarditis will develop The remainder, either as a result of recurrent bouts of rheumatism and reinfec-

tions of the heart, or possibly because of the inherent nature of the original infection with its subsequent chronic progression and contraction without any recognized reinfection, will develop signs of mitral stenosis or aortic stenosis at some future time

When this same patient whose progress we are following does not develop mitral stenosis but persists in manifesting a moderately loud systolic murmur while well and ambulatory, he may be considered as having organic mitral insufficiency This is especially true if there is pulsation or dilatation of the left auricle or general cardiac hypertrophy It has been maintained by certain authors that organic mitral insufficiency does not exist or is extremely rare, and that in cases in which such diagnoses are made there is either no disease of the mitral valve or there is mitral stenosis This point of view was a reaction against the previously prevailing belief that every patient with a systolic murmur had mitral insufficiency We now know that in many such cases there is no structural disease of the mitral valve They were often instances of nervous or hyperdynamic hearts with a benign systolic murmur or cases of myocardial failure of the degenerative type with a relative mitral insufficiency but with no endocarditis Another group of patients at postmortem examination showed mitral stenosis The conclusion was drawn especially from postmortem data that organic mitral insufficiency did not exist without stenosis The difficulty with this point of view is that we cannot deny the presence of a disease which is generally nonfatal by the use of autopsy data It would be just as fallacious to maintain that acute tonsillitis and chickenpox are extremely rare because one almost never finds cases at autopsy So it is with organic mitral insufficiency This condition generally develops into mitral stenosis, which eventually is fatal, although for many years only an incompetency of the valve is present As long as it is only a regurgitation, the patient is apt to be in good health Even this is not invariably so for some patients die from heart failure showing a markedly dilated heart and mitral insufficiency without stenosis Only occasionally have we an opportunity of examining the valve before stenosis develops, e g , if subacute bacterial endocarditis becomes superimposed on mitral insufficiency Then we see that the past history of rheumatism and the moderately loud systolic murmur indicated a true rheumatic mitral endocarditis, producing a regurgitation but no stenosis of the valve, for the pathologic examination will show both the old rheumatic and the recent bacterial lesions Furthermore, during life many cases of mitral insufficiency without stenosis show a definite systolic expansile pulsation of the left auricle on fluoroscopic examination, which finding affords further evidence of regurgitation through the mitral valve The conclusion from the fore going is that, although the diagnosis of organic mitral insufficiency should be made with caution, it is a condition that actually exists, especially in young rheumatic individuals

MITRAL STENOSIS

Let us now discuss the development of the signs of mitral stenosis How soon the changes to be described will occur is a variable matter

Upon rare occasions they begin within one year, although generally many years elapse between the original infection and the development of definite evidence of mitral stenosis During these years, the symptoms may be none or few, such as slight dyspnea and palpitation If we examine the patient from year to year, little change may be detected until finally, as the first indication that the mitral valve is becoming stenosed, we commence to detect a snapping quality or accentuation of the first heart sound as heard at the apex During these same years the pulmonary second sound may have become somewhat accentuated and reduplicated As an aid in diagnosis, I have found the quality of the pulmonary second sound of distinctly less value than the quality of the first sound In other words, an accentuation of the first sound is frequently the first suspicious evidence of early mitral stenosis This, however, is not sufficient to enable one to make a definite diagnosis, for there are other conditions in which it occurs, such as hyperthyroidism, a short P-R interval, anemia, cardiac neurosis *and some cases of hypertension*

When this snapping quality of the first heart sound is heard, it is well to listen carefully to the heart in order to hear the early development of presystolic or mid diastolic murmurs At this time no murmur whatever may be heard during diastole on ordinary examination, but if one listens directly after a brief effort or with the patient in the left lateral position, in some cases a presystolic murmur may thereby become audible which otherwise might be entirely overlooked I have frequently brought out this very important physical sign which made a definite diagnosis of mitral stenosis possible when, under the ordinary examination, it was entirely inaudible This typical rumble which is quite pathognomonic of mitral stenosis may also be uncovered by the use of the *amyl nitrite* test Here the patient inhales amyl nitrite for a few seconds and one auscults carefully during the acceleration of the heart that follows the inhalation In practice, it is sufficient to listen at the apex with the patient in the left lateral position or directly after a brief period of exercise, e g , twenty-five hops Whether a diastolic or presystolic murmur is heard or not, frequently a short sharp sound may be audible in early diastole at the apex This is called the opening snap and is strongly suggestive of mitral stenosis In many cases it is also followed by a rumbling murmur, but at times the latter only becomes clear months or years later When the presystolic or mid diastolic murmur is heard, no matter how it is brought out, and the first sound is snapping in quality, the patient may be considered to have mitral stenosis Just as is true of most useful rules in medicine there are always exceptions I recall examining a boy 12 years old in 1939 who showed a definite mid-diastolic murmur When he was placed in the left lateral position a clear presystolic murmur became audible In addition there was an apical grade II systolic murmur I believed he had definite mitral stenosis Seven years later when I examined this boy quite carefully I could hear no murmurs whatever, even in the left lateral position, and was of the impression that he had no valvular lesion

At some later time it will be found that the same mid diastolic or pre-

systolic murmur will be audible even with the patient at rest or lying recumbent It will be much better heard with the patient in the recumbent than in the upright position Furthermore, the murmur may be quite sharply localized over a very small area in the region of the apex As years go on and the degree of stenosis of the mitral valve increases, the presystolic or late diastolic murmur lengthens so that the time is reached when almost the entire diastolic pause is filled with the murmur The early portion of this long rumbling murmur may have a diminuendo quality and the latter part a crescendo element, terminating in the accentuated first sound

At this time we are not considering the development of generalized circulatory failure, which may come at any time during the patient's progress, but which generally does not occur until the mitral stenosis is well advanced In the meantime the heart has been essentially regular in its rhythm, although there may have occurred occasional extrasystoles either of ventricular or auricular origin The dominant rhythm, however, is likely to have been regular Some time during the life of this patient who has already developed mitral stenosis auricular fibrillation is apt to develop This complication is very rare during the first decade, becomes more frequent the next decade and is most common about the ages of 35 or 40 Whereas the heart during all these previous years was regular, the beat now becomes tumultuous and grossly irregular This change is sudden when it occurs, so that on one day a normal slow heart rhythm may have been present and on the following day it may be found to be very rapid and absolutely irregular Not infrequently this auricular fibrillation is transient and recurs in the form of attacks Each attack may last several hours and then disappear the heart returning to its slow regular rate Some weeks, months or years later the same phenomenon may be repeated During the attack, the patient may suffer with a good deal of palpitation, dyspnea and general nervous agitation If the state of the reserve strength of the circulation is not great before the attack the clinical condition of the patient may become quite serious Edema of the lungs, cyanosis and orthopnea may develop When an attack of auricular fibrillation with a rapid ventricular rate produces no evidence of cardiac failure, it denotes a satisfactory state of myocardial reserve After a variable number of transitory attacks of auricular fibrillation the condition is apt to become permanent In fact, in most cases, once the heart becomes grossly irregular, it remains so indefinitely

It is most important to be able to recognize this type of irregularity, first because it is extremely common, and second because much in the way of treatment can be done for it Furthermore, a proper understanding of this phenomenon aids greatly in comprehending the changes that take place when it occurs as evidenced in the physical examination of the heart When auricular fibrillation develops the auricles cease contracting, remain distended in diastole and show fine fibrillary twitchings throughout the musculature The ventricles on the other hand begin to contract rapidly and very irregularly In auricular fibrillation, there is an extremely large number of impulses traversing the auricles The number is in the vicinity of four hundred or more Only a small proportion of these impulses succeed in

reaching the ventricles, the remainder being blocked at the junctional tissue. In other words, the conduction apparatus between the auricles and ventricles (the auriculoventricular node and bundle of His) is unable to transmit such a large number of irregular impulses from the auricles and only a third or so get through. The result is that the ventricles contract irregularly at the rate of about one hundred and twenty to one hundred and fifty. These contractions are grossly irregular. The pulse is also irregular, both in time and in force, and there is very apt to be an appreciable pulse deficit, i. e., the pulse rate as counted at the wrist will be distinctly less than the heart rate at the apex.

It is not difficult to recognize this condition on bedside examination, using only the apparatus that we carry with us in general practice at all times. A rather simple and fairly satisfactory rule of thumb may be described as follows. Given a patient who obviously has heart disease, who shows a heart rate as counted at the apex of over 100 and has a pulse rate that is distinctly less (a pulse deficit of 10 or more), if the rhythm appears to be grossly irregular the condition is auricular fibrillation nine times out of ten. There are rare exceptions in which this rule would not apply. One would feel even more certain of the diagnosis of this arrhythmia under the above circumstances if it were known that the patient had mitral stenosis *and a history of rheumatic fever*. The *triad of rheumatic fever, mitral* stenosis and auricular fibrillation is so frequently found in the same individual that as a practical matter, knowing that two exist, the third should be suspected and be sought for.

There is one final point which may be helpful in detecting auricular fibrillation at bedside examination when there still remains some doubt in the diagnosis. Occasionally, numerous extrasystoles coming quite irregularly or from different foci of the heart may produce such a tumultuous rhythm, even with an appreciable pulse deficit, that it would be difficult to distinguish it from auricular fibrillation. One important distinction between the two conditions, however, will be noted on auscultation. In both conditions quick beats and long pauses seem to be coming irregularly, resulting in sudden acceleration and sudden retardation of the heart beat. When the condition is due to extrasystoles, every time there is a long pause, it is a compensatory mechanism and therefore follows a previous quick beat. The same succession of long pauses after quick beats also obtains in auricular fibrillation. The phenomenon that distinguishes the two, however, is the appearance of a long pause that is *not* preceded by a quick beat, for this occurs only in auricular fibrillation and not with extrasystoles. In other words, it is important to auscult carefully over the apex of the heart for a sufficient time to detect a sudden lengthening of the heart cycle and to recall whether the previous cycle was a short one or a long one. If a cycle of average length or one of longer duration is followed by a long pause, the condition is due to auricular fibrillation. Furthermore, the quality and intensity of the first heart sound vary much less with cycles of different length than is the case when the irregularity is due to extrasystoles. In addition to the foregoing observations, whenever it is possible

to do so an exercise test may help in diagnosis When the rate accelerates in this manner, extrasystoles generally disappear and the irregularity of auricular fibrillation becomes more prominent

Apart from the bedside method of diagnosis, auricular fibrillation is easily diagnosed by the use of graphic measures, such as the polygraph or the electrocardiograph After a mechanical registration of the venous pulse is made one obtains certain waves, two of which are primarily the result of the contraction of the right auricle and the left ventricle, respectively The contraction of the right auricle sends an impulse upward through the vena cava and produces a slight wave in the jugular vein The contraction of the left ventricle sends an impulse upward through the aorta and produces a wave in the carotid artery The first is called the *a* wave and the second, the *c* wave When auricular fibrillation is present, the auricles are no longer contracting, and, therefore, the *a* wave disappears Likewise, in the electrocardiograms the representative of auricular contraction (the P wave) disappears In its place there may be found numerous fibrillary waves (*f* waves) running more or less throughout the heart cycle These small waves represent the numerous fibrillary twitchings that are going on in the auricles in patients with this condition In Chapter 21 these matters are taken up in greater detail At this point, it may be stated that whereas cardiography gives the final proof of the diagnosis of auricular fibrillation, it is possible in the vast majority of cases to make accurate diagnoses by the use of simple bedside methods

Let us now consider the effect of auricular fibrillation on the physical signs in a patient who has mitral stenosis Before doing so it is necessary to review briefly the process involving the flow of blood from auricles to ventricles As the ventricle begins to relax and dilate in diastole after the previous systole blood flows from the left auricle into the left ventricle merely as a result of differences in pressure and gravity The pressure in the ventricle starts from zero at a time when the left auricle has already received considerable blood from the lungs and has developed some pressure In fact, normally about seven eighths of the blood flows from the left auricle to the left ventricle through the mitral valve by this mechanism Only the last bit of blood is propelled by the contraction of the auricle which gives the ventricle its final stretching just before it contracts It follows that the early and middle portions of the diastolic murmur of mitral stenosis are produced independently of auricular contraction and it has been proved convincingly that only the presystolic portion is produced by auricular systole Now, if auricular fibrillation develops in the patient we are considering, who had a murmur practically filling diastole, that portion of the murmur due to auricular systole will disappear because in auricular fibrillation the auricles cease contracting In actual practice this is just what happens

If, therefore, we examine the patient on one day when the heart is regular and find a murmur completely filling diastole we should observe on the following day, if auricular fibrillation is present that the presystolic interval is now clear and that the murmur extends only through the first portion of

the diastole It may be difficult or impossible during the early days after this change has occurred to appreciate the actual disappearance of the presystolic murmur This is so because the heart rate is still rapid When the rate is rapid, the diastolic pauses are short and they do not permit the silent portion of the diastolic interval to appear Each first heart sound comes so soon after the previous second sound that, although the early portion of the diastolic murmur alone is present, it actually extends throughout the short diastole and gives one the impression that the presystolic murmur is still present If, however, one listens carefully when the heart rate is slower, one finds that the diastolic murmur has remained unchanged When the pauses are long enough and the following first heart sounds are sufficiently removed from the previous cycle, nothing whatever will be heard in presystole, i e , the presystolic murmur has disappeared with the development of auricular fibrillation This discussion may seem complicated, but it is *of some importance and deserves emphasis* The *presystolic murmur* by definition has its constant relationship with the following *first* heart sound, no matter how long the diastolic pause may be If mitral stenosis exists and there is a normal mechanism, toward the end of the diastolic pause a rumble will often be heard The earlier and mid-diastolic portion of the murmur of mitral stenosis is not due to auricular systole and has its constant relationship with the previous *second* heart sound It is that portion alone that persists when auricular fibrillation is present

It is possible for auricular fibrillation to develop in a patient with mitral stenosis at any time, even when only the presystolic murmur had previously been present The degree of stenosis may not be sufficient or the dynamics may be such that the velocity of the flow of blood from left auricle to left ventricle is too slow to produce a rumble One can readily see that if this occurred we might lose the only murmur in diastole that existed The presystolic murmur would disappear entirely because the auricles were no longer contracting and there was no other murmur in diastole I have seen such cases in which the diagnoses were properly made and proved to be correct at postmortem examination These, of course, are rare experiences In one case, the patient was still thought to have mitral stenosis despite the absence of any murmur in diastole because on x-ray examination the left auricle was unusually prominent and the electrocardiograms showed right *ventricular preponderance* These additional aids in the diagnosis of mitral stenosis will be taken up in greater detail below In general, there are two types of cases of mitral stenosis when a murmur in diastole may be very faint or entirely inaudible In the first type the stenosis is very slight and the state of the circulation very efficient In the second type the stenosis is moderate or marked, but the degree of heart failure is considerable The preceding discussion and particularly the experience just cited sufficiently emphasize the importance of the relation between auricular fibrillation and the murmurs of mitral stenosis

I have paid considerable attention to auscultatory findings in mitral stenosis because I regard them to be of primary importance One may gain the impression that a great deal of overemphasis is being attached

to the value of the stethoscope in this whole matter To be sure, one obtains the most satisfactory appraisal of the degree of cardiac embarrassment from a brief inspection of the patient The history of the symptoms of circulatory insufficiency, the degree of dyspnea, the distention of the cervical veins and the amount of passive congestion in the body give one much more important information concerning the state of the circulation than the determination of the presence or absence of a diastolic murmur However, in the vast majority of cases the final decision as to whether the patient has or has not mitral stenosis will depend upon auscultation Symptoms and peripheral signs of congestion tell us whether or not a patient has *heart failure* but auscultation tells us whether or not there is *valvular disease* Patients have valvular disease for many years before and for a longer time than they have heart failure In fact, during the early years before cardiac insufficiency develops, without most careful auscultation it will frequently be impossible to tell whether the patient has organic heart disease or no heart disease

There are other criteria that are useful in the diagnosis of mitral stenosis The finding of a definite thrill in the apex region that is diastolic in time is quite reliable evidence of the diagnosis of mitral stenosis When the heart rate is not rapid, the proper timing of this thrill is not difficult When the rate is 100 or over, it is almost impossible to distinguish the thrill that occurs in diastole from the vibration that is produced in a hyperactive heart Such vibrations which are really systolic in time may be felt in patients with hyperthyroidism, in those who are in the acute stages of rheumatic infection and in those with certain nervous states I have frequently observed instances in which such vibrations were misinterpreted and considered to be diastolic thrills with the result that mistaken diagnoses of mitral stenosis were made It has been my general experience that when the detection of the diastolic thrill could be made with certainty, auscultation would easily confirm the diagnosis, whereas in many cases in which the diagnosis of mitral stenosis was quite certain, thrills were entirely absent The finding of the diastolic thrill, however, may have greater importance when there is a combined lesion of the mitral and aortic valves

When hypertension is absent the volume of the pulse in mitral stenosis is customarily small (pulsus parvus) Cyanosis of the lips, cheeks and other portions of the face is common in mitral stenosis Even when actual cyanosis is not present such patients are apt to have a rather florid countenance This is in contrast to the pallor that typifies aortic disease Mitral stenosis is also more common in the female than in the male Another feature that characterizes mitral stenosis in contrast to disease of the aortic valve is its chronicity Patients with mitral stenosis may continue to show cardiac failure over a great many years with repeated intervals of improvement It is possible for such a patient to have congestive heart failure and be alive and in fair comfort five to ten years later This does not occur with anything like the same frequency in aortic cases, for here the life expectancy is very short once decompensation develops Hoarseness from pressure on

the left main bronchus and dysphagia from pressure on the esophagus may result from enlargement of the left auricle Rarely aphonia and paralysis of the left vocal cord occur from pressure of the dilated pulmonary artery on the recurrent laryngeal nerve Pulmonary infarctions occur in mitral stenosis because of a tendency for the formation of mural thrombi within the cavities of the auricles, particularly in the auricular appendages when auricular fibrillation is present From these thrombi, which may remain silent for many years, emboli may be dislodged and if they come from the right auricle they produce infarction of the lung, while if they come from the left auricle they produce peripheral emboli in the greater circulation with resultant hemiplegia, or other embolic complications Pulmonary infarction is by no means always due to cardiac emboli, for it is a common finding in chronic congestion of the lungs no matter what the cause may be, and can result from local thrombosis of the pulmonary vessels or from thrombosis of the veins of the pelvis or leg *Hemoptysis* is also common in mitral stenosis apart from its occurrence as a result of pulmonary infarction Such hemoptysis may be quite brisk, the patient raising a mouthful or more than a cupful of bright red blood This may bring to one s mind the possibility of pulmonary tuberculosis or bronchogenic carcinoma, both of which conditions need to be searched for in doubtful cases It is striking how clear the lungs may be shortly after an attack of hemoptysis in mitral stenosis, as if the bleeding resulted from a rupture of a small dilated vessel and did not reflect a high degree of generalized passive congestion In some ways the hemoptysis resembles nosebleeds that occur in rheumatic individuals

Considerable light has been thrown on this type of hemoptysis by Dock It has been found by injection experiments that the bronchial veins can be enormously dilated in cases of mitral stenosis, whereas normally they are very small and difficult to visualize They resemble miniature varices similar to those found in the esophagus in cases of cirrhosis of the liver This mechanism does not apply to the cases in which bloody sputum is raised as a result of pulmonary infarction or in those in which there is acute pulmonary edema In the former the sputum is blood stained or contains blood clots and in the latter pink, forthy sputum appears, while in the condition now being considered there is considerable pure blood in the absence of much obvious pulmonary congestion

X-ray and Electrocardiography

There are times when the diagnosis of mitral stenosis is doubtful, even after the most careful bedside examination There remain two means at our disposal which may further aid in this regard, i e, x ray and electrocardiography As a result of stenosis of the mitral valve, the pressure in the left auricle increases and this chamber dilates This dilatation may be apparent on an x ray film of the heart exposed in the ordinary manner A bulge may be seen in the left upper border just below the pulmonary artery Sometimes it is quite prominent Occasionally it is difficult to distinguish this, except by fluoroscopic examination, from the dilatation of the pulmonary artery that occurs with patent ductus arteriosus or other conditions

Furthermore, the left auricle may extend with undue prominence posteriorly and bulge into the posterior mediastinal space On rare occasions the left auricle will be so dilated that it will actually extend across the midline and form the right upper border of the heart The large left auricle may produce a definite angulation of the esophagus and push it to the right This can be observed on fluoroscopic examination or on a roentgenogram taken while a barium meal is being swallowed Even the left primary bronchus may be seen on x-ray examination to be raised upward or constricted by a dilated left auricle These observations may sometimes be extremely helpful in distinguishing mitral stenosis from congenital heart disease Finally with improved x ray technic, it has been possible to see calcification of the mitral valve on fluoroscopic examination and less frequently on the flat x ray plate When this is properly done it always means mitral stenosis and it can be distinguished from calcification of the annulus fibrosus which usually causes no disturbance in the function of the heart and is an unimportant finding Rytand has called attention to an auscultatory sign that might lead one to suspect the presence of a calcified mitral annulus It consists of detecting a short apical diastolic murmur in elderly patients with or without a systolic murmur who also have heart block The sound is due to auricular activity The detection of calcification of valves has already proved of great aid in diagnosis, especially when combined lesions are present

The electrocardiogram occasionally gives helpful indirect evidence of mitral stenosis As a result of hypertrophy and dilatation of the auricles in this condition, the auricular complex (P wave) may develop a peculiar form, become unduly large prominent, notched and have a flat top (see Chap 21, Figs 128 129) When these changes are marked they are almost invariably indicative of mitral stenosis A further change that results from mitral stenosis is hypertrophy of the right ventricle The pulmonary pressure is increased and with it a compensatory hypertrophy and dilatation of the right ventricle take place Inasmuch as the electrocardiograms give some measure of preponderant hypertrophy of one ventricle over another in mitral stenosis they may show certain alterations from the normal These changes consist of prominent downward deflection of the initial ventricular complex in Lead I and a prominent upward deflection in Lead III In addition there are changes in the precordial leads that are even more valuable in detecting hypertrophy of either ventricle Such curves however, must be interpreted with great caution for not infrequently they may be obtained in subjects with emphysema or in patients with certain forms of congenital heart disease Electrocardiography in general has not been very valuable as an aid in valvular diagnosis, but occasionally this evidence coupled with other data which of themselves might not have been sufficient enables one to make a proper diagnosis

Common Signs of Heart Failure

Throughout this discussion nothing has been said about the common signs of heart failure that may be found in a patient with mitral stenosis,

such as engorged liver and cervical veins, generalized edema or enlargement of the heart These are not particularly characteristic of mitral stenosis although it must be stated that enlargement of the heart in mitral stenosis is more apt to be transverse, especially to the right, than it is in other forms of heart disease The signs of heart failure associated with mitral stenosis will be taken up in detail when discussing the problem of general cardiac failure

Little has been said about the presence of a systolic murmur at the apex in mitral stenosis This, of course, is a very common finding but does not indicate that the mitral valve is stenosed It means that there is a concomitant mitral insufficiency or it has the questionable significance that accompanies systolic murmurs in general It has been generally taught that mitral stenosis is almost always associated with mitral insufficiency Whether or not regurgitation of blood actually takes place in many of these cases is difficult to ascertain The only physical evidence of this would be the presence of an apical systolic murmur Now, it is not sufficiently appreciated that, in a large number of cases of clear cut mitral stenosis, no systolic murmur whatever can be heard The same may be said of stenosis of the aortic valve I am of the opinion that mitral stenosis without any insufficiency is very common, and that it often develops in patients who previously never had a systolic murmur

It also must be understood that mitral stenosis is not always the sole valvular lesion of the heart There may be additional involvement of the aortic or more rarely of the tricuspid valve The diagnosis of combined lesions is no simple matter, but in so far as it can be determined, it will depend upon the proper utilization of the criteria for each individual lesion with some regard for the effect of one upon the other

Mitral Stenosis and Blood Pressure

This discussion would not be complete without some mention of the relation between mitral stenosis and the blood pressure The old term *pulsus parvus* reflects the general impression that the pulse is small and the blood pressure is low in this condition In a study of almost 800 cases of mitral stenosis, it was found that when the disease was present in young people, the blood pressure tended to be lower than that of the average population of that age group However, in patients with mitral stenosis who were in the higher decades of life the average blood pressure increased decidedly until it reached 180 systolic and 95 diastolic for patients 60 to 69 years of age This I feel cannot be an accidental finding, for certainly no group of patients suffering from some unrelated condition, such as cancer of the breast or ulcer of the stomach, has hypertension as a general rule It is hard to believe that mitral stenosis itself accounts for the hypertension for when present in young persons, the pressure is even lower than normal Yet how are we to explain the frequent association of mitral stenosis and hypertension?

One may at the outset maintain that the mitral stenosis in these older

hypertensive patients is not rheumatic in origin and that it is due to sclerotic changes in the mitral ring similar to the peripheral arterial sclerosis with which the hypertension is associated One might object to the accuracy of the diagnosis of mitral stenosis in these elderly hypertensive persons on the ground that the presystolic gallop rhythm which is common in hypertension might be mistaken for the presystolic murmur of mitral stenosis In answer to the latter it may be said that the diagnosis in a sufficient number of these elderly patients has been confirmed at autopsy As far as the former criticism is concerned, apart from the general view that I have expressed before, in which it was stated that mitral stenosis is probably due to no other cause than rheumatism, it was found that 50 per cent of these hypertensive patients had a past history of rheumatic fever or chorea, which percentage corresponds fairly closely to that of a rheumatic history in any group of patients having mitral stenosis Inasmuch as mitral stenosis per se does not produce hypertension, it is possible that the underlying chronic rheumatic infection insidiously produces arterial changes that terminate in hypertension Another possibility is that a long standing disease process in the heart by some reflex mechanism can initiate hypertension Furthermore, the great frequency of hypertension in the older patients with mitral stenosis may be due to the fact that the elevation of the blood pressure enabled them to survive and reach the later decades of life A more likely explanation it seems is that mitral stenosis and hypertension are commonly associated because the underlying rheumatic infection has a predilection for patients who have a vascular vulnerability The disease has a tendency to select certain types of people and this so-called vascular type is vulnerable both to the infectious and to the degenerative form of heart disease The frequent association of rheumatic heart disease and angina pectoris in the same family is additional evidence in favor of this conception

There is also some clinical and statistical evidence to indicate that the hypertension developing in patients with mitral stenosis need not produce any deleterious effect, but on the contrary may be actually helpful Although it may add certain symptoms as a result of the hypertension itself, when these are not severe, there is reason to believe that it delays the progress of mitral stenosis It is very curious that so many patients with mitral stenosis over 50 years of age have hypertension in addition I believe that the hypertension prolongs their lives and enables them to reach the later decades, for the great majority of patients with mitral stenosis without hypertension die before they reach the age of 50 One may explain the beneficial effect of hypertension by supposing that as a result the cavity of the left ventricle tends to remain large and somewhat dilated and that this dilatation tends to counteract or delay the antagonistic progressive contraction that is going on in the mitral ring as a result of the rheumatic mitral stenosis Another possible factor is that mitral stenosis is a burden on the right side of the heart and hypertension embarrasses the left side of the heart In this way the two burdens are equalized and if congestive failure

is due to an imbalance of the two ventricles, as is believed by many authorities, the effect may be beneficial I have frequently seen elderly patients with mitral stenosis and hypertension continue to do fairly well many years after it was expected that they would die It therefore appears to be a somewhat favorable sign to find an increasing blood pressure in cases of mitral stenosis

4

Diseases of the Aortic and Tricuspid Valves

There are certain striking differences between diseases of the aortic and the mitral valves Aortic valve disease is more common in males than in females and the reverse is true for mitral disease Pain in the chest of the type that is characteristic of angina pectoris is frequent in aortic disease and is rare in mitral disease Pallor is more typical in the former, whereas plethora or at least a florid countenance is more typical in the latter Patients with aortic valve disease, although frequently complaining of chest pain and palpitation, have much less dyspnea as compared with those having mitral disease They generally also have better bodily strength and remain ambulatory and able to do work for a longer time with outspoken evidence of valve disease, however when general circulatory insufficiency does develop, with dyspnea congestion of the lungs and liver or peripheral edema they have not the recuperative power that is commonly seen in patients with mitral stenosis In other words, when decompensation is once developed in an aortic case the outlook is rather grave The average length of life after such an occurrence is not apt to be more than two or three years *In mitral cases on the other hand frequently several attacks of de-*compensation occur, each followed by a partial restoration of circulatory efficiency The patients are thereby enabled to continue for many years on restricted activities Pulmonary infarctions are less common in aortic than in mitral cases This may be partly but not entirely accounted for by the fact that auricular fibrillation is extremely rare in disease of the aortic valve and very common in mitral stenosis Disturbances in conduction, such as heart block and bundle branch block, are much more common in aortic than in mitral disease The same is also true of attacks of syncope and sudden death A final difference that has impressed me in recent years is that subacute bacterial endocarditis is more common in aortic disease than in mitral stenosis This does not mean that the mitral valve is less frequently involved in subacute bacterial endocarditis, for in most cases when the mitral valve is involved there is only an insufficiency of the valve but no stenosis

There are three general causes of disease of the aortic valve namely, rheumatism, syphilis and arteriosclerosis The young are apt to be rheumatic, the middle aged, syphilitic and the aged, hypertensive or sclerotic This division is quite arbitrary as many cases overlap from one age period

into another The frequency with which one type or another will be met will depend in a great measure on the locality where the physician praticces In certain sections of the country almost all cases or aortic insufficiency will be luetic in origin This discrepancy has made some authors state that aortic insufficiency is invariably due to syphilis In a locality like New England, more individual cases of rheumatic aortic insufficiency are seen than any other type, although frequently this is associated with other valvular disease On rare occasions aortic insufficiency may be functional or transient This may occur during the very asthenic stages of marked anemia of the pernicious type and in hypertension

AORTIC INSUFFICIENCY

The two types of lesions of the aortic valve to be considered are aortic insufficiency and aortic stenosis Let us first take up aortic insufficiency The heart in this condition will become hypertrophied in the course of time and in some cases this enlargement becomes extreme The left ventricle is the chamber that bears the main brunt of the leak and therefore it becomes not only thickened but eventually considerably dilated This enlargement extends downward more than outward so that the apex impulse may be felt in the sixth interspace outside the nipple line or even lower The apex impulse is apt to be heaving and forceful denoting a thickened musculature of the left ventricle It is well to become familiar with this type of impulse no matter under what circumstances it is felt for it gives a fairly satisfactory indication that the wall of the left ventricle is thickened and sometimes enables one to decide that the heart is hypertrophied, even when percussion outlines are doubtful One gains the impression that with systole the apex impulse lifts the palpating finger and keeps it elevated for an appreciable time before it recedes It does not merely tap the finger Only on rare occasions can a diastolic thrill be felt at the base of the heart in aortic insufficiency Percussion merely aids in estimating whether the heart outline is enlarged or not, and in no way helps in identifying aortic valvular disease Auscultation reveals the decisive evidence of the diagnosis In uncomplicated conditions, the heart rhythm will generally be found regular Almost invariably a systolic murmur will be heard at the base of the heart and often there is a systolic murmur at the apex as well The apical systolic murmur is either due to a relative mitral insufficiency or to a concomitant mitral endocarditis When the aortic insufficiency is due to syphilis or sclerosis the apical systolic murmur should be regarded as due to relative mitral insufficiency and not to a true endocarditis because a true organic mitral insufficiency practically never results from syphilis or arteriosclerosis Here the left ventricle becomes markedly dilated and the mitral ring, although essentially normal, is large and the leaflets do not completely close the opening during systole When it is rheumatic in origin, it will always be difficult to decide whether or not the mitral valve is structurally diseased unless additional evidence is obtained pointing to mitral stenosis as well If aortic stenosis is also present an apical systolic murmur may actually be coming from the aortic valve

There are three possible explanations of the basal systolic murmur in aortic insufficiency First, it may be due to an actual structural or relative stenosis of the aortic valve This I think is more common than is recognized Secondly, it has been thought that a roughening of the aortic wall such as is found in syphilis and arteriosclerosis may produce a systolic murmur This I think is fallacious, for I have frequently heard no murmur when later at autopsy the aorta showed extreme degeneration Finally, it may be due to the hyperdynamics of the circulation at that moment There may be a momentary increase in the rate of flow of the blood during systole that accompanies aortic insufficiency I believe that the latter factor is more important than is generally assumed

The apical and basal systolic murmurs discussed in the foregoing paragraphs, although generally present in aortic insufficiency, are in no way diagnostic, for they occur when the aortic valves are competent The most important and characteristic finding is the presence of a diastolic murmur heard at the aortic area, and even with louder intensity at the third left interspace near the sternum and propagated downward This murmur has a blowing quality, starts directly with or replaces the second heart sound and is diminuendo in character At times it is quite faint and requires most careful auscultation for its detection In some cases it will be audible only if searched for with the patient sitting up and examined during deep expiration In general it is unwise to make the diagnosis of aortic insufficiency without this finding

Although the detection of an aortic diastolic murmur is valid evidence of aortic incompetency, especially if there are peripheral signs as well, postmortem examination may occasionally fail to confirm the finding of valvular disease This does not mean that the diagnosis was incorrect, for the valve may have been incompetent in life and still show no structural disease at autopsy When the heart is examined post mortem we do not see the structures as they existed with their normal tone and under blood pressure relations that prevailed during life The valves may have been relatively insufficient because of dilatation of the aortic ring Such relative aortic insufficiency is not uncommon in hypertension and in marked anemia when the valves are structurally sound Similar dynamic dilatation has been repeatedly ob erved in the aorta itself I have seen instances in which the x ray showed very marked dilatation of the aorta, pointing strongly to the diagnosis of aneurysm, when at autopsy a perfectly normal small elastic aorta was found

There are instances in which a presystolic murmur will be heard at the apex in cases of aortic insufficiency This is the so called Austin Flint murmur It may be difficult to interpret the significance of an Austin Flint murmur, for it has the same time and quality and is heard in the same place as the murmur of mitral stenosis It must be admitted, however that this murmur can be present when mitral stenosis is absent The practical point of view that I have taken in this regard is that if the so-called Austin Flint murmur is heard in syphilitic aortic insufficiency, I would accept it as real, but that when it is present in a rheumatic case, I would be apt to

regard it as due to concomitant mitral stenosis This is based on the empirical pathologic experience that syphilis never produces mitral stenosis and that rheumatism frequently does, even when aortic disease is also present However, I have seen instances of rheumatic aortic insufficiency in which not only a presystolic murmur was present at the apex but also a presystolic thrill and x-ray evidence of a prominent left auricle were noted in cases in which at postmortem examination mitral stenosis was not demonstrated

Peripheral Signs

There are, in addition, very important peripheral signs of aortic insufficiency which are more or less dependent upon the increased pulse pressure that accompanies this condition The most striking one of these is the hyperactive agitated pulsations of the arteries, especially the carotid arteries They beat violently so that the entire lateral portions of the neck seem to pulsate The radial pulse has a water-hammer quality, the so called Corrigan pulse There is a capillary pulse that may be made out on observing the fingernails while exerting mild pressure This may also be detected in watching a flush come and go after rubbing the forehead or while pressing on the inside of the lower lip or the lower lobe of the ear with a glass slide In addition, there is the so called "pistol shot in the femoral artery that one may detect with the stethoscope Here, if pressure is applied during auscultation, a systolic murmur may be heard This murmur is really a normal phenomenon, but what is more significant is that in many cases of aortic insufficiency with appropriate pressure over the femoral artery, one may hear a distinct diastolic murmur (Duroziez's sign) These various peripheral evidences of aortic insufficiency are by no means pathognomonic They occur in a less well marked degree in a variety of conditions Many states in which there is a lax atonic peripheral vascular tree may present these signs They are commonly seen in association with hyperthyroidism, certain types of anemia, especially pernicious anemia, fevers, nervous hearts, and in some cases of hypertension In most of these conditions the pulse pressure will also be found greater than normal I have frequently observed the capillary pulse and a Corrigan pulse in conjunction with the above conditions, sometimes even noting pulsation of the retinal arteries

The peripheral signs of aortic insufficiency, although in general not so helpful in diagnosis as the diastolic murmur, nevertheless are valuable when there are combined valvular lesions This is especially true in rheumatic aortic insufficiency Here, it may be difficult to interpret the origin of a diastolic murmur heard along the left sternal border Such a murmur may have various causes At times the diastolic murmur of mitral stenosis may be so prominent and loud that it is actually well heard even toward the base of the heart The diastolic murmur, on the other hand, may be due to an aortic insufficiency, which is combined with the mitral stenosis It may be a so-called ' Graham Steell murmur This latter murmur is supposed to be due to a relative pulmonary insufficiency occurring in mitral stenosis, and is heard in the pulmonary area (second left interspace) and alontheg

left sternal border Moreover, the presence of a diastolic murmur in this area together with the systolic murmur that is also present may be attributed to congenital heart disease Finally it can be the result of organic tricuspid stenosis Frequently it is impossible to make an accurate differentiation In such instances the peripheral signs may decide the question as to whether aortic insufficiency is present or not One may have to utilize all available means of diagnosis to arrive at a proper decision The presence of auricular fibrillation will point definitely to mitral stenosis and the snapping quality of the first heart sound should make one suspect its presence The x-ray finding of a dilated pulmonary artery will point to the diagnosis of a patent ductus arteriosus When the patient has obvious aortic insufficiency and the question of an additional mitral stenosis comes up, if auricular fibrillation exists, both valves are probably involved Also, if there is right ventricular preponderance in the electrocardiograms or a prominent left auricle on x ray examination, it is likely that the lesion is not confined to the aortic valve, but that mitral stenosis is also present, for if aortic insufficiency were the sole lesion, we would expect marked left ventricular preponderance in the electrocardiograms Although this evidence is not indisputable, it is helpful In order to make the diagnosis of a Graham Steell murmur, it would be necessary to find this diastolic murmur present in the absence of peripheral signs of aortic insufficiency One would also expect that the patient would be decompensated and that the murmur should either disappear entirely or at least diminish markedly in intensity as improvement in the circulation occurred Although in most cases the interpretation of the basal diastolic murmur is not difficult when it is doubtful it is apt to be of academic interest, for it may be assumed that except in extremely rare occasions a diastolic murmur means heart disease

AORTIC STENOSIS

Stenosis of the aortic valve is frequently due to rheumatic infection, is occasionally the result of arteriosclerosis, but is never syphilitic in origin There was a time when the diagnosis of aortic stenosis was made much too frequently, as many patients who merely had a basal systolic murmur were regarded as suffering from this condition This was followed by a period of undue caution when it was regarded as a very rare disease At present we must take a middle course for there is ample experience to teach us that it is by no means rare and that it is frequently overlooked

The younger individuals with aortic stenosis are generally regarded as suffering from rheumatic valvular disease, whether the aortic lesion is found to be an isolated one or is combined with mitral or tricuspid involvement Individuals with aortic stenosis who are over 50 or 60 years of age, who frequently show no significant involvement of the other valves, have been regarded as arteriosclerotic It has been my impression that many, if not most, of such patients are also rheumatic It is curious that even in this older group a positive history of previous rheumatic infection will be elicited in almost 30 to 40 per cent of the cases This is true notwithstanding the fact that the interval between the possible early infection and the present

illness might have been forty to fifty years, that a history of rheumatism may easily have been forgotten, and that many years ago atypical rheumatic infections were doubtless overlooked. Furthermore, as a result of a better understanding of the pathologic findings in rheumatism, pathologists are now willing to admit that cases which they formerly had called arteriosclerotic they are now ready to call rheumatic. Another argument against the arteriosclerotic origin of many of these cases is that as a rule the ascending aorta, where sclerotic processes in general are so extensive, is apt to show comparatively few atheromatous changes in aortic stenosis. This may, however, be due to a protective effect that the stenosed valve exercises in buffering the systolic impact of the aorta. It is also conceivable that some cases of aortic stenosis are the result of a previous nonrheumatic infection of the aortic valve, or may even represent the healed stage of a previous subacute bacterial endocarditis. Finally, the likelihood that arteriosclerosis alone can be responsible for a portion of the older cases cannot be entirely dismissed.

The general clinical features of aortic stenosis resemble those of aortic insufficiency in that it is more common in males, it is found in hearts with a regular rather than an absolutely irregular rhythm and is rarely associated with emboli or pulmonary infarction. The left ventricle is often enormously hypertrophied, with only very little dilatation. The heaviest hearts are those with aortic stenosis, although the largest silhouettes on x-ray examination are those of mitral stenosis. This is so because of the marked dilatation that occurs with the latter condition. Dyspnea comes late, the patients maintaining rather good health for long periods of time until congestive failure finally intervenes. When this occurs, the prognosis is grave, for death is apt to intervene within two or three years. The foregoing generalizations do not hold, however, when there is combined mitral and aortic valvular disease.

One frequent complication of aortic stenosis is angina pectoris. This, I believe, is often overlooked. It has currently been taught that angina is a common accompaniment of aortic insufficiency and little mention is made of its association with other valvular lesions. The explanation given is that the heart is nourished in diastole, for during the actual systolic contraction blood flows out through the aorta while the coronary arteries are practically occluded. In aortic insufficiency blood is regurgitating back to the left ventricle from the aorta during diastole and with the diminished diastolic pressure there is inadequate nourishment of the heart muscle. Although this seems to be a logical explanation, there are certain conflicting clinical facts. In patients with aortic insufficiency the occurrence or severity of angina by no means follows the level of the diastolic pressure. Furthermore, in cases of syphilitic aortic insufficiency in which the mouths of the coronary arteries are not occluded the degree of regurgitation is very often of an extreme degree and yet angina is rare. On the other hand, it has seemed to me that in patients with aortic valvular disease and angina pectoris the element of stenosis has been more prominent than that of regurgitation.

In fact, I have frequently seen angina with the former when there was no evidence of the latter

The explanation of the frequency of angina in aortic stenosis is obscure Possibly several factors are involved It is now believed that there is coronary flow during systole as well as diastole The ventricular wall is generally thicker in aortic stenosis than in pure aortic insufficiency or in other types of heart disease This may result in a greater degree of relative myocardial anoxemia because the coronary blood supply may not keep pace with the hypertrophy, and oxygen diffusion into thick muscle fibers is more difficult Furthermore, the work of the heart must be enormously increased to expel blood through such narrow valves Also the velocity of the blood ejected through the stenosed valve must be terrific to maintain a normal output per minute This rapid stream may possibly exercise a suction action on the mouths of the coronary arteries which lie just beyond the valve If this happens, blood actually is extracted from, rather than fed to, the heart during systole Finally, numerous cases have additional significant coronary sclerosis to account for the angina However, I have seen young patients with aortic stenosis and angina in whom sudden death occurred, and postmortem examination showed normal coronary arteries Here some of the above mechanisms may have been involved

Angina is frequently overlooked in aortic stenosis because insufficient attention is paid to minor complaints such as mild sternal distress on hurrying Often the patient will not even mention it to his physician and it will have to be brought out by direct questioning This accounts for the frequent occurrence of sudden and unexpected death that is so common in aortic valvular disease Granted that an integral part of the symptom complex called angina pectoris is a likelihood of sudden death, eliciting a proper history will often forewarn the physician of possible sudden fatalities in aortic cases that otherwise would remain unexplained Furthermore, when no such previous history of angina can be obtained it is not inconceivable that a first attack of angina may end fatally Not so long ago I saw a man of 55 who was suffering from congestive heart failure After his condition had improved it was planned that a complete thyroidectomy would be performed Many other physicians and I had seen him over a period of a year and we all were in accord in the diagnosis of aortic stenosis Finally, on direct questioning, I found that he had a definite history of anginal distress of about two years duration, a fact that he never mentioned before because his primary complaint was breathlessness The day before the time set for the operation, while he was apparently in good condition, he suddenly expired If the diagnosis of angina had not been made this patient s demise would have been regarded as one of those unexplained sudden deaths in aortic disease Experiences similar to this one are not uncommon

Another peculiarity of aortic stenosis is that it is frequently accompanied by a tendency to faintness or to actual syncope The exact explanation of this is obscure but it may be linked up with peculiarities in the carotid

sinus reflex It is now well known that if this reflex is hyperactive, giddiness, weakness and fainting attacks can occur It is, therefore, necessary to study the condition of this reflex in cases of aortic stenosis in the hope of throwing further light on this otherwise obscure phenomenon So far, however, in most cases a normal sensitivity of the carotid sinus has been found Syncope occurs particularly on effort and in the last analysis is probably due to cerebral anoxemia It is unlikely that it is due to temporary heart block, despite the fact that conduction defects are common in aortic stenosis May it be due to a temporary increase in heart rate with a decrease in cardiac output? The problem is still unsolved

In this connection it is of interest that in aortic stenosis the heart rate is often slow This may be so even when gross evidence of congestive heart failure is present In fact there are few conditions apart from aortic stenosis in which advanced congestive heart failure will be found with a heart rate under 70 or 60, in which the slow rate cannot be accounted for by heart block or the administration of digitalis This point has been useful in first directing suspicion to the diagnosis of aortic stenosis and then detecting *the direct clinical evidence of the lesion by careful examination* This slow heart rate may possibly be due to the same factor that makes the patient subject to syncope or may in some way be related to the vagus apparatus

For some unknown reason, the basal metabolism may be elevated in some cases of aortic stenosis This may occasionally occur when there is little or no evidence of congestive failure The increase in metabolism may be as great as 20 to 40 per cent I have seen several proved instances of aortic stenosis in which repeated examinations showed such readings and yet on gross and microscopic study of the thyroid gland nothing abnormal was found The explanation that the increase is due to the marked cardiac hypertrophy hardly seems valid as many other cardiac patients with great enlargement of the heart have shown a normal metabolism This problem needs serious consideration when the question of latent thyrocardiac disease comes up

The clinical diagnosis of aortic stenosis will in many cases depend on finding a systolic thrill at the base of the heart When it is marked it can readily be felt in the second right interspace or over the upper or middle portion of the sternum It is often missed because careful palpation is not practiced or because it is not searched for properly A fainter thrill may only become apparent if palpation is performed during a held expiration with the patient sitting upright or leaning forward It can be confused with systolic thrills that accompany congenital heart disease, but the differential diagnosis is generally established without difficulty by the presence or absence of other findings such as cyanosis, clubbing of the fingers, electro cardiographic changes (right ventricular preponderance with pulmonary stenosis and left preponderance with aortic stenosis) and x ray examination One must be careful to interpret as a thrill only that condition in which a real purr of significant duration is felt In this way it will not be mistaken for systolic or other impacts of the chest that are common in hyperactive hearts or in thin chested individuals This differentiation is

important, for true basal systolic thrills in the absence of congenital heart disease generally mean aortic stenosis

A second indisputable sign of aortic stenosis is the finding of calcification of the aortic valve on x ray examination This is best detected by fluoroscopic examination but may even appear on the flat heart plate It needs to be distinguished from calcification in the mitral valve or in the myocardium This can be easily done by a trained observer, the main differential points being its location and the fact that at the very beginning of systole calcification in the mitral valve will be seen to move upward whereas the aortic valve moves downward It is obvious that calcification of a valve must be a late process, and when it is sufficiently advanced to become visible the valve must have been diseased for years In fact, in many cases in which calcification is not very extensive it will not be detected It is surprising, however, how frequently this diagnosis was made on x ray examination and in all such cases that were examined post mortem the x ray diagnosis was confirmed Although calcified aortic stenosis is generally seen in older people it is not at all rare in younger persons In the younger group the disease is obviously rheumatic in origin and I suspect that in many of the older group it is also of rheumatic source although the older patients have been generally regarded as arteriosclerotic It must be borne in mind that calcium deposits are often found in any long standing chronic inflammatory process and if a patient survives an aortic lesion long enough calcification may result

The other clinical signs of aortic stenosis are less important because they are more difficult to interpret The second aortic sound is diminished in intensity or absent in many cases The difficulty is that the pulmonary second sound may be audible in the aortic area making it impossible to distinguish the origin of these sounds A systolic murmur will be present in practically every case of aortic stenosis This murmur is best heard in second right intercostal space (the aortic area) or not infrequently over the upper precordium and midsternal region It is generally a fairly loud murmur and coarse in quality The difficulty is that basal systolic murmurs are common in such a variety of circumstances that this alone cannot constitute reliable evidence for the diagnosis of aortic stenosis When it is appreciated however, that the constricting process, which eventually becomes sufficient to produce a palpable systolic thrill or calcification of the valve detectable by x ray, is a very slow one extending over many years it is evident that the diagnosis can only be suspected in many instances during these intervening years When phonocardiograms are taken the classical systolic murmur has a peculiar diamond shaped appearance (Fig 188) Although this is not pathognomonic it is sufficiently characteristic to distinguish it occasionally from the murmur of mitral insufficiency It is obvious that a very loud murmur must have been fainter or even very slight years before or at its onset I am convinced that many patients who merely show systolic murmurs of moderate or loud intensity without much other evidence of heart disease have aortic stenosis, even when the systolic murmur is louder at the apex than at the base of the heart I have followed such cases long

enough to see the true nature of the lesion develop At first a systolic murmur would be heard when there were no symptoms Such a patient may have been refused insurance because of it At this time a thrill, a diastolic murmur or appreciable hypertrophy would be absent The murmurs might have been called functional systolic murmurs I might have made the diagnosis of mitral insufficiency, because in some of these cases the murmur was quite prominent near the nipple as well as toward the base of the heart, or the murmurs may have been designated as instances of systolic murmurs, cause unknown' Then as years went on a definite basal thrill would become palpable or calcification of the valve would be found on x-ray examination Dyspnea or anginal pain were frequent eventual developments in such patients

Although most cases of aortic stenosis in which the diagnosis can be made will have moderate or very loud murmurs (grade III to VI), there will be some with only faint basal murmurs I recall a man I followed for twenty years who always showed a grade I (rarely grade II) aortic systolic and diastolic murmur He had had an early history of rheumatic fever and remained quite well all these years He had a fairly large chest with an increased anteroposterior diameter Although the murmurs were very faint, calcified aortic stenosis could be seen fluoroscopically, which was confirmed eventually post mortem In such cases the systolic murmur is faintly heard because of the thickness of the intervening space between the heart and the outside of the chest

The lesson to be drawn from these experiences is that we should be more ready to make the diagnosis of aortic stenosis before all the classical signs are present

Insufficiency so frequently accompanies stenosis of the aortic valve that one should make a careful search for a basal diastolic murmur When it is found it will lend support to the diagnosis that the aortic valve is at least diseased and that the systolic murmur, which can be due to so many other causes, is in fact the result of aortic valvular disease Even the presence of a systolic and diastolic murmur at the aortic area does not necessarily indicate stenosis of the valve Both murmurs are present in most instances of free aortic regurgitation without stenosis such as occurs in syphilitic aortic insufficiency, a condition which never results in stenosis of the valve It is sometimes taught that when there is stenosis of a valve there must be insufficiency, i e , if a valve is so deformed and scarred that it cannot open it would similarly be impossible to close completely Although this view sounds logical and one is further impressed by its validity on seeing the rigid calcified valves post mortem, the fact remains that in many instances of aortic stenosis no diastolic murmur will be audible nor will there be any peripheral signs of aortic insufficiency The same holds true for mitral stenosis, for here in many instances, although there may be a long diastolic rumble indicating a fair degree of stenosis, no systolic murmurs will be heard after the snapping first heart sound

There are other auscultatory signs in aortic stenosis Murmurs may be present at the apex of the heart indicating an additional organic involvement

of the mitral valve or a relative insufficiency of that valve The basal systolic murmur and thrill may be transmitted upward into the vessels of the neck The finding of a systolic murmur and thrill over the vessels of the neck, without similar signs over the chest, cannot be relied upon as certain evidence of aortic stenosis, because they may be present in other conditions such as hyperthyroidism, anemia and nervous states Furthermore the apical systolic murmur may merely be transmitted from the aortic valve and may not signify any additional lesion

The plateau form of radial pulse is fairly characteristic of aortic stenosis It is often overlooked because the examiner does not think about it When it is present the pulse will be found to rise and remain sustained longer than in other conditions It will obviously be absent if there is also sufficient aortic insufficiency, for this will counteract the plateau character with a collapsing quality

The blood pressure in aortic stenosis is variable In the classical case the systolic level is apt to be low and the diastolic comparatively high, producing a small pulse pressure One frequently finds readings such as 110 mm systolic and 90 mm diastolic Two other factors, however, affect the blood pressure so that almost any reading might be found in different cases, i e, an accompanying aortic insufficiency and an independent essential hyper tension The result is that both the systolic and the diastolic pressure may vary from very low to very high levels

The x-ray in typical aortic stenosis will show a prominent, rounded left ventricle producing a boot shaped heart The electrocardiogram will reflect the degree of left ventricular preponderant hypertrophy that is present and occasionally disturbances in conduction

DISEASE OF THE TRICUSPID VALVE

Although functional insufficiency of the tricuspid valve is common, organic disease of this valve is rare It is easy to understand the frequency with which this valve becomes relatively incompetent When there is con gestive failure, the heart is generally dilated If the right ventricle dilates, the tricuspid ring is thereby stretched and the valve although normal in structure will no longer be able to close the enlarged opening This will necessarily result in a regurgitation of blood from the right ventricle into the right auricle This condition is common in all types of heart disease, but particularly in mitral stenosis where the burden on the right side of the heart eventually becomes great Under such circumstances, the patient is apt to have not only marked evidence of cardiac failure with cyanosis, edema and engorged liver, but regurgitation of blood through the tricuspid valve may show itself in other ways There may be an actual systolic pulsa tion of the veins of the neck and limbs, and a pulsating liver It is no simple matter to detect these changes, for normally there are pulsations in the jugular veins which are difficult to time Likewise it is difficult to distinguish systolic pulsations of the liver from pulsations of the right side of the heart and abdominal aorta By placing both hands around the lower right axilla and right upper quadrant of the abdomen, one may at times be

able to sense an actual systolic expansion in the liver region with each heart beat as one hand moves forward while the other moves backward In many cases the significance of a systolic pulsation is misinterpreted because it can be due to an impact from the neighboring abdominal aorta or the overlying heart The most satisfactory clinical evidence of tricuspid regurgitation is the detection of faint pulsations in the veins of the forearms or forehead As a result of tricuspid insufficiency, there is also a systolic murmur heard in the tricuspid area over the lower sternum It is very difficult to identify this murmur or to distinguish it from the systolic murmur of mitral origin heard at the apex that is invariably present and has the same quality It is thought that having the patient hold a deep breath may accentuate the systolic murmur if tricuspid insufficiency is present It probably is true that many patients with advanced cardiac failure have a functional tricuspid insufficiency even when no such diagnosis is made, for frequently at postmortem examination the tricuspid ring is dilated

True tricuspid endocarditis is practically never a pure lesion It has the same etiologic background as disease of the mitral valve and in my experience it has invariably been associated with mitral stenosis with or without aortic stenosis The diagnosis of organic tricuspid insufficiency or tricuspid stenosis is extremely difficult Many authorities have considered it impossible to make this diagnosis However, with the recent increased interest in this condition, more and more cases are being recognized There are certain features which, although not pathognomonic, should arouse suspicion of the presence of tricuspid stenosis

Patients with tricuspid stenosis have in general the same symptoms as those with mitral stenosis Cyanosis in this condition is apt to be more intense, infarctions of the lung are very common and the liver is invariably enlarged A striking feature in some of the cases is the fact that the liver may remain enlarged for a long time, even years, while the patient is comparatively comfortable and ambulatory One common and fairly characteristic observation is the rapid change in the appearance of the face when the patient changes from an upright to a recumbent position Within a minute or so the face, which appeared normal or merely showed a very slight tinge of cyanosis, becomes markedly suffused and intensely cyanotic The veins of the neck and head will also become very distended This change probably results from the fact that on assuming the horizontal position the increased venous return to the right side of the heart cannot be delivered quickly through the constricted tricuspid valve and backs up in the veins For similar reasons this sign ought to be present in cases of constrictive pericarditis

Ascites may also be very prominent and require frequent abdominal tappings The same thing may be true of hydrothorax, especially on the right side Patients with tricuspid stenosis seem to tolerate these evidences of advanced heart failure with less dyspnea and general distress than do patients with other cardiac conditions It is thought by some observers that in the course of time these patients are apt to develop a peculiar

appearance of the skin, consisting of a slight olive discoloration I have seen this in several instances and it has seemed to be a mild chronic jaundice with a slight faint greenish tint to it It probably is the result of prolonged enlargement of the liver and no doubt may develop in other cardiac patients when the same long continued stasis takes place Polycythemia is also an occasional finding in tricuspid stenosis Abdominal symptoms are common, particularly pain and tenderness in the liver What is more impressive, however, is the degree of comfort and the comparative absence of dyspnea and peripheral edema in the presence of a persistently enlarged liver and recurrent ascites To this extent it resembles constrictive pericarditis which is distinguished from tricuspid disease by the absence of significant enlargement of the heart and murmurs

On physical examination the cervical veins are almost always found to be distended and prominent These characteristics may occur in all types of right heart failure The distinguishing feature in tricuspid cases is that the condition is not only very marked but it persists for long periods of time, even when the patient is in a fairly good state of compensation The same will be true of the venous pressure It will not only be elevated, but with improvement it will not return so closely to the normal level as in other cardiac diseases with the exception of constrictive pericarditis The blood pressure is practically never significantly elevated in tricuspid stenosis (over 150 mm systolic) Hypoproteinemia is common, probably the result of prolonged hepatic insufficiency

Enlargement of the right side of the heart is very striking The right auricle dilates tremendously and may even extend beyond the right mid clavicular line There is almost invariably a right ventricular preponderance in the electrocardiograms Auricular fibrillation, although often associated with tricuspid stenosis is not so frequent with the same degree of heart failure as in cases of mitral stenosis alone This means that a fair number of these patients will show gross heart failure with a regular rhythm The murmurs are similar to those that occur in mitral stenosis, but they are well heard over the lower sternum or to the right of the midline When it is appreciated that in a series of thirty-two cases of tricuspid stenosis every case also showed well marked mitral stenosis, difficulties in the interpretation of murmurs become apparent When the systolic murmur and particularly the diastolic murmur are better heard near the midline than at the apex of the heart, it would lead one to suspect that the tricuspid valve is diseased The frequent association of tricuspid stenosis with mitral stenosis, aortic stenosis and even adhesive pericarditis makes accurate diagnosis extremely difficult

It is of considerable interest that among patients with chronic rheumatic valvular disease, those with tricuspid stenosis die at the earliest age The age at death in these cases (34 years) is about twelve years less than in those with mitral stenosis alone (46 years) and nineteen years less than in those with aortic stenosis alone (53 years) However, the duration of symptoms or evidence of congestive failure is much greater in patients with tricuspid disease than in those in whom the other valves are involved A recent

study has shown comparative figures to be seven and five tenths years for tricuspid stenosis, four and six-tenths years for mitral stenosis, and three and one-tenth years for aortic stenosis In other words, although death occurs at the youngest age in the tricuspid cases, the patients with this disorder live longest once failure develops This is because they have a mechanical embarrassment of the heart comparable to constrictive pericarditis, but the constriction is inside the heart at the tricuspid valve, and not outside in the pericardium This process is more static and does not necessarily imply heart muscle failure We, therefore, are witnessing in such cases evidence of right-sided failure, such as venous distention, enlarged liver and ascites, which has essentially a mechanical cause Such congestion would obviously be much less serious or progressive than if it resulted from pure heart muscle failure

It follows that if a case of known mitral stenosis carries on fairly well for years with evidence of right heart failure and shows persistently engorged cervical veins and enlarged liver, especially if this is out of proportion to the degree of dyspnea, and there is no hypertension, the diagnosis of tricuspid stenosis must be considered

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Diseases of the Pericardium

Disease of the pericardium is almost always secondary to some other primary condition, whether it be an infection or some other morbid process. The pericardial abnormality may impair health or threaten life in one of several methods. As part of an inflammatory process, pus may form and give rise to the hazards attendant to a closed empyema. Sterile fluid, an exudate, a transudate or a hemorrhage may be so abundant that it produces cardiac tamponade, i.e., interference with the normal movements of the heart. Finally, as a result of a previous infection or some other pathologic conditions, scars, adhesions or bands may form in the pericardium impeding the free contraction or expansion of the heart. This latter process can conveniently be divided into two clinical types: *chronic constrictive pericarditis* and *nonconstrictive pericarditis*, the latter embracing the condition generally called *chronic mediastinopericarditis*.

The various common types of acute pericarditis will first be discussed, then pericardial effusion, and finally, chronic constrictive and nonconstrictive pericarditis will be considered.

RHEUMATIC PERICARDITIS

Acute rheumatic pericarditis generally develops about two weeks after the onset of the rheumatic infection. Its occurrence, however, is variable. At times it ushers in the rheumatic infection, actually preceding any other manifestation of rheumatism. In fact, it may be the only evidence of the rheumatic infection, there being no joint pains whatever. On the other hand, it can develop after the original attack of rheumatism has subsided, just as one is ready to allow the patient to get out of bed. It is more commonly associated with aortic disease than with mitral stenosis. This may be explained when we consider the anatomic relation between the pericardium and the valves of the heart. At the base of the heart there is only the thickness of the aortic wall between the visceral pericardium and the aortic valves, while the entire musculature of the ventricles intervenes between the visceral pericardium and the mitral valve. If the process of infection is one of extension no matter in which direction, it is easily conceived how pericarditis would be more often associated with disease of the aortic than of the mitral valve.

Symptoms

The symptoms of acute rheumatic pericarditis consist essentially of an accentuation of those symptoms that already exist as a result of the underlying rheumatic process The heart, already rapid, becomes further accelerated so that a rate of 130 or more is not at all unusual The respiratory rate becomes unduly rapid Although this need not be associated with actual dyspnea, in some instances the rate of respiration may be 50 and the patient may be able to lie flat in bed In most cases, however, there is actually some respiratory distress The marked increase in the heart and respiratory rates is out of proportion to the degree of fever which often is no more than 101 or 102 F Very frequently there is a rather peculiar and characteristic cough This is short, hacking, irritative and unproductive Often there is pain in the chest It was formerly thought that this chest pain was due to the inflammation of the pericardium, but there is now considerable doubt about this explanation There are frequent painless instances of acute rheumatic pericarditis Certain studies on human beings indicate that the pericardium for the most part is insensitive to pain It may be, therefore, that the pain in pericarditis is due to an associated pleuritis Notwithstanding the different possible explanations, pain in the heart region is fairly *common in pericarditis Occasionally the early pain is in the abdomen* rather than over the heart and with the fever and leukocytosis that accompany it, an acute surgical condition of the abdomen may be simulated

Diagnosis

The diagnosis may be suspected from the evidence described in the preceding paragraph, but will finally rest upon detecting the to and fro pericardial friction rub This is generally a harsh, grating sound, best heard in the third or fourth left interspace It can remain localized or spread over a larger area On auscultation, the sounds seem to be close to the ear and the intensity may be augmented by firm pressure with the stethoscope or by having the patient bend forward Thus to and fro friction on rare occasions is quite transient, lasting only some hours, but generally it persists for days When it is loud, it seems to envelop the heart sounds rather than to follow them When it is typical, it has two portions to it, one with systole and one with diastole, but I have seen instances in which for about twenty-four hours there was only a systolic element and later the diastolic portion was heard At times, the continuity of the sounds is interrupted so that it may give one the impression that there are three or four portions to the entire friction rub Because of the frequent association with aortic insufficiency, which has a to and fro systolic and diastolic murmur, it is sometimes quite difficult to distinguish a to and fro pericardial friction rub from the signs of aortic insufficiency In fact, both conditions may be present In some cases, it is necessary to delay judgment If the to and fro murmur disappears, it must obviously have been due to a pericarditis If it persists indefinitely, it is due to aortic insufficiency The detection and proper interpretation of a pericardial friction rub is most important because it may

be the only distinctive evidence that there has been an acute pericarditis

There is another frequent development in acute rheumatic pericarditis that is helpful in diagnosis, particularly if by chance the pericardial friction was either missed or indistinct This is the so called Ewart s sign It consists of dullness and bronchial breathing heard below the angle of the left scapula These findings are supposed to be due to atelectasis and compression of the left lower lobe of the lung from fluid accumulating in the posterior pericardial sac It is not certain whether this explanation is true but it is clear that the above signs which resemble pure lobar pneumonia frequently occur in acute rheumatic pericarditis The bronchial breathing in this condition is apt to be quite loud, and generally is unassociated with any rales The resemblance to pneumonia accounts for the frequent history obtained from rheumatic patients that they had pneumonia in childhood I have seen numerous such instances in which the physician thought the child was suffering from pneumonia Had not the proper diagnosis been made such patients would have presented themselves later with aortic insufficiency in whom no other infection than pneumonia could be blamed as the cause of the valve disease The development of Ewart s sign generally follows by some days the appearance of the pericardial friction rub It may, however, actually precede it I recall an experience in which such a case was shown as an instance of pure lobar pneumonia in a ward given up solely to the care of pneumonia patients Because the patient had some of the secondary features of rheumatism discussed in Chapter 2, namely, repeated nosebleeds and a family history of rheumatic heart disease, and because he already had some aches in his joints, it was predicted that a pericardial friction would appear and other features pointing to rheumatic infection would develop This occurred during the following days I cite this case to illustrate the importance of accurate diagnosis and the indirect means by which proper diagnoses are sometimes made

The third characteristic of rheumatic pericarditis is the frequent development of disturbances in conduction Heart block is very common in this condition When actual blocking of beats occurs, it is easily detected by auscultation The rhythm which is rapid and regular will suddenly be interrupted by pauses These pauses will not be preceded by a premature beat, which distinguishes it from extrasystoles A complete heart cycle every now and then actually drops out When the disturbance in conduction is less marked, no actual blocking of beats occurs The heart remains perfectly regular It is almost impossible to detect this change without graphic methods, although the presence of a gallop rhythm or a decrease in intensity of the first heart sound arouses one s suspicion If an electrocardiogram or a polygram is taken, the conduction time as indicated in these curves will be found above the upper limits of normal which is 0 2 sec These slight changes are earlier evidence of what becomes heart block when the process is more marked Disturbances in conduction indicate a simultaneous myocarditis which frequently accompanies pericarditis Except for occasional cases of coronary thrombosis, this is the only form of pericarditis in which conduction disturbances are apt to occur Apart from the

friction sound, the Ewart's sign and the conduction disturbances, there is little that is distinctive on physical examination in cases of rheumatic pericarditis

It is likely that in most cases of rheumatic pericarditis an effusion of a greater or lesser extent develops When it is slight, it is impossible to detect it The inflammation gradually subsides and the fluid is absorbed The pericardial friction disappears after lasting a few days, but the signs of compression of the left lower lobe of the lung and the conduction disturbance may persist for two weeks or more Gradually both of these signs also disappear, although on rare occasions the electrocardiograms may continue to show a delayed conduction time indefinitely Changes in the ventricular complexes of the electrocardiograms may also be present (Chap 21, Fig 171)

Prognosis

After a prolonged illness, generally lasting months, the patient recovers The prognosis in rheumatic pericarditis in general is good The immediate mortality is about 15 per cent The physician should always keep before him the possibility and even the likelihood of a favorable outcome in the face of what seems to be a very stormy disease One must appreciate that we are dealing *primarily with an infection which at this time is in an acute* fulminating stage and that the acute process can subside Patients may be desperately sick and make an excellent recovery even if signs of congestive heart failure develop Acute rheumatic pericarditis practically never leads to constrictive pericarditis

The ultimate outcome, if immediate recovery takes place, will depend in a great measure upon whether the valves have been affected during the acute process If during the attack of pericarditis no diastolic murmur develops, recovery may be complete from a symptomatic point of view The presence of a systolic murmur will have the same significance as systolic murmurs in general This matter is discussed in detail in other chapters I have seen numerous instances in which, after a most violent and desperate attack of rheumatic pericarditis, recovery was complete and the patient was subsequently able to carry on even strenuous physical work In such cases there may even be no evidence of heart disease whatever in later years The subsequent health of the patient and ability to work are quite different when evidence of valve disease develops Such experiences have impressed upon me most forcefully the importance of the valves in heart disease Recovery from injury to the myocardium after acute infections seems to take place most satisfactorily, but when the valves are structurally involved, in many cases there appears to start a progressive vicious cycle, with subsequent development of circulatory insufficiency

Great emphasis has rightly been placed on the importance of the myocardium in heart failure This applies primarily to nonvalvular heart disease In chronic rheumatic heart disease a fairly healthy ventricular musculature is noted on postmortem examination If the myocardium rather than the valves were of primary importance in rheumatism, it is surprising that we

do not see patients in later life, who have had rheumatic fever in childhood, dying of heart muscle failure without valvular disease Apart from the fatalities that occur during the acute rheumatic carditis when the state of the myocardium is all important, death due to heart damage in subsequent years is limited to those patients who develop valvular lesions, while those who recover from the acute infection without murmurs or without valvular injury may never have any further trouble from the heart The degree of valve deformity is not the sole final cause of failure of the circulation, although it is a most important factor in the disability, the so called acci dents of heart disease intervene, such as bacterial endocarditis, infarctions, emboli and infections When these accidents do not occur, then the valvular damage present at the time of heart failure will be found to be adequate to explain the situation without recourse to any significant role played by the heart muscle

Treatment

The treatment for a patient with rheumatic pericarditis comprises little more than that employed for the underlying rheumatic fever It is custom ary to give salicylates either by mouth or by rectum as has been described in a former chapter Some believe that large doses of salicylates actually produce absorption of the pericardial effusion Digitalis will have no beneficial effect unless there is evidence of congestive heart failure The patient, of course should be made as comfortable in bed as possible and requires careful nursing to spare him any unnecessary effort He generally will find it more comfortable to be in the semirecumbent or at times in the upright position Codeine in doses of $\frac{1}{4}$ to $\frac{1}{2}$ grain may be given every four hours even to children, for precordial pain or cough The only additional therapeutic measure that may be helpful is the use of an icebag over the precordium This sometimes alleviates the heart pain and diminishes the sensa tion of palpitation In only a very rare case will it be necessary to tap the pericardium The fluid then will generally be serous but occasionally may be hemorrhagic Tapping the pericardium may be a life saving measure and will be taken up under the general discussion of pericardial effusion

No doubt in the near future endocrine therapy will be of value in this as in other rheumatic conditions Compound E (cortisone) and ACTH have already produced astounding and prompt improvement in severe cases of rheumatic pericarditis

PERICARDITIS IN PNEUMONIA

There are certain distinctive features in the pericarditis that occurs with pneumonia In the first place, it is not a common complication of pneu monia Secondly it occurs almost exclusively in those cases of pneumonia that have empyema of the pleura Furthermore it is more commonly asso ciated with empyema in the left pleural cavity than in the right In fact, if a diagnosis of pericarditis is made in a patient with pneumonia, empyema of the pleura, especially on the left side, should also be sought for Pericarditis when it does occur, is apt to develop in the latter days or after the

crisis or lysis would be expected A most important difference between the pericarditis in pneumonia and in rheumatic fever is that the pneumococcus is likely to produce pus and rheumatic fever never produces pus For this reason the diagnosis of the former is important

The diagnosis of pericarditis with pneumonia will rest almost entirely upon detecting the pericardial friction rub When this occurs and is heard, its interpretation is a good deal more simple than in rheumatic fever, for there are no endocardial murmurs to confuse the picture If the pericardial friction rub is not heard, pericarditis may yet be suspected in occasional cases of pneumonia if during the second week of the disease there develops a left pleural empyema which is satisfactorily treated and yet a septic course continues One must remember that a patient suffering from pneumonia may have an empyema cavity in the pleura, well cared for, and yet die of an empyema of the pericardium If there is any suspicion of this, an exploratory puncture of the pericardium is indicated because the effusion under such circumstances is purulent and not likely to heal spontaneously If pus is found in the pericardium, surgical drainage should be instituted This complication of pneumonia is a most serious one The outlook is practically hopeless if the pericardium is not drained although recovery may take place under surgical treatment

Since the introduction of sulfonamide and penicillin therapy pneumococcus pericarditis, which formerly was uncommon, has become extremely rare However, it is not unlikely that unrecognized subclinical cases are occurring and recovering without surgical drainage May not some of these cases form the background upon which subsequent constrictive pericarditis will develop?

VIRUS PERICARDITIS

With increasing interest in and prevalence of virus infections it has become evident that some cases of acute pericarditis hitherto regarded as of unknown origin and probably in many instances called rheumatic are, in fact, due to a virus infection This may be a complication of a virus pneumonia or may occur without any significant involvement of the lung parenchyma There may be a concomitant pleuritis with or without pleural effusion In these cases the customary pericardial friction sound and other findings previously described including possible changes in the electrocardiograms will be found Because of the precordial pain, pericardial friction, slight fever and abnormalities in the electrocardiograms the condition may readily be confused with acute coronary thrombosis These cases do not show evidence of valvular involvement and whatever effusions occur are never purulent The course of the illness may be polycyclic with remissions and reappearance of the pericardial friction and even a new pericardial effusion I have seen instances in which three such cycles took place over the course of a few months The prognosis is generally favorable and complete recovery is to be expected Treatment is symptomatic unless preparations like aureomycin will prove effective

TUBERCULOUS PERICARDITIS

Tuberculous pericarditis is very rare in general practice Its mechanism and development are quite similar to tuberculous pleurisy In both conditions the process may be dry or fibrinous, or a serous or serosanguineous effusion may be poured out and adhesions may develop Tuberculous pericarditis, however, is a much more serious complication of tuberculosis It is always secondary to tuberculosis of the pleurae, lungs or mediastinal glands The diagnosis of this condition in the early stages will depend almost entirely on the detection of a to and fro pericardial friction rub as in other types of acute pericarditis There will be no evidence of either heart muscle disease or of valvular disease, for the heart proper is very refractory to the tuberculous infection The striking characteristic of tuberculous pericarditis is a tendency to large pericardial effusions and it is the one condition that may require repeated tappings The exudate will be found to be sterile on ordinary bacteriologic examination, just as occurs in rheumatic effusions Here, on the other hand, inoculating some of the fluid into a guinea pig may prove that it is tuberculous or occasionally the tubercle bacilli may be found on staining part of the fluid or on culture Whenever a large effusion is found (800 to 1200 cc) especially if there is a tendency for reaccumulation, tuberculosis should be suspected Effusions are apt to be larger than in rheumatism because they are poured out slowly, allowing the heart and pericardial sac gradually to accommodate themselves to the increased pressure, and because the heart itself is not diseased In rheumatic pericarditis, the reverse is true The accumulation of the fluid is more rapid and either the heart muscle or the valves are generally affected The heart then would have failed before such extensive effusions as occur in tuberculosis could have been poured out

It is quite likely that many cases of tuberculous pericarditis are unrecognized, run a mild course and heal thereby forming the basis of chronic constrictive pericarditis in later years In some instances evidence of constrictive pericarditis may develop within a few months after the first detection of acute pericarditis There may not be a large pericardial effusion and what fluid there is may be pocketed and not free

Although in the past there was no specific treatment for this condition except for the occasional need for pericardial tap now streptomycin or similar antibiotics would be indicated In fact, successful use of instillations of streptomycin into the pericardial cavity have been reported

PERICARDITIS WITH CHRONIC NEPHRITIS

Pericarditis is a very common complication of chronic nephritis Its exact nature is not altogether clear Although it may be due to a terminal infection, in some cases bacteriologic examination of the pericardium and pericardial fluid at autopsy shows no evidence of any infectious organisms In other cases, streptococci are found More likely it is purely vascular in origin and similar in its mechanism to the ulcerations in the bowel that are frequently found in cases of uremia It develops insidiously in those

who have advanced chronic nephritis with marked nitrogen retention There frequently is a slight fever and leukocytosis with the pericarditis These need not be present The disease may be entirely painless The diagnosis will rest on the finding of the pericardial friction rub It is only important from the point of view of prognosis for it denotes quite decidedly that the end is near Out of twenty four cases of pericarditis and nephritic that I once studied, in twenty three instances death occurred within three weeks The other patient was practically moribund when he was last seen More recently one patient survived four months after the friction was first detected Pericarditis sometimes develops in an ambulatory patient who has advanced chronic nephritis and even here it indicates that a fatality is to be expected within one month There is no therapy whatever that can be offered for this condition, for it is the underlying nephritis with its uremic manifestation that is the real problem While following a patient with advanced chronic nephritis, it is important to watch for acute pericarditis purely as an aid in estimating the prognosis, for sometimes such information is needed in giving certain advice to the family and patient

PERICARDITIS WITH CORONARY THROMBOSIS

A more detailed discussion of coronary thrombosis will be taken up later (Chap 6) At this point the single feature of pericarditis which frequently develops in coronary thrombosis needs to be considered Following an attack of partial or complete occlusion of a coronary artery, the part of the ventricles supplied by this vessel becomes infarcted When the process of infarction is sufficient to extend from within the ventricle to its surface, the pericardium is necessarily involved At this point, a localized fibrinous exudate develops and therefore a true serofibrinous pericarditis will result It is obvious that if the infarction does not extend to the visceral pericardium, no pericarditis occurs, or if the site of the lesion is in the posterior part of the heart or over the dome of the diaphragm even if the pericardium is involved a friction rub might not be audible Furthermore, the friction rub that does develop with coronary thrombosis can be very faint and transient, and, therefore, can easily go undetected This finding will be more common the more frequently and the more carefully patients with coronary thrombosis are examined, but will not be detected in more than 10 to 20 per cent of the cases

Generally the friction rub becomes audible in one to several days after the onset of the attack Sometimes it appears later when the patient is already free from the agonizing pain that was present in the early days It does not add any additional symptoms to what the patient already has and it does not appreciably alter the prognosis of the condition or the treatment, except in so far as it indicates a significant area of myocardial infarction It is a helpful sign in making the diagnosis in some doubtful cases The fact that there is a fever and a leukocytosis does not mean that an infection of the pericardium is taking place, for these result from the original infarction of the heart muscle and may be regarded as a reaction to the

absorption of foreign protein Only on very rare occasions is pericardial effusion associated with this type of pericarditis

MISCELLANEOUS FORMS OF PERICARDITIS

We have just discussed the six most common conditions in which inflammation of the pericardium takes place There remains a heterogeneous group of conditions in which the pericardium may become the site of inflammation or of the accumulation of fluid Any generalized sepsis may localize in the pericardium It is then likely to be a terminal event There is one type, not extremely rare, in which pericarditis due to streptococcus infection occurs with the development of a purulent effusion This really is a part of a more general streptococcus infection and is especially associated with an antecedent sore throat There are also instances of acute pericarditis that may be called idiopathic for they do not fall into any of the forementioned groups They present the picture of an acute infection, possibly virus in origin, do not produce empyema, show no stigmata of rheumatism, but may resemble acute coronary thrombosis because of the chest pain and the presence of minor electrocardiographic changes This condition runs a favorable course and recovery is complete This has been covered in the discussion of virus pericarditis Furthermore, in any cachectic state such as advanced carcinoma leukemia or pernicious anemia, when the red blood cell count is extremely low, fluid may accumulate in the pericardial sac as it does in other cavities of the body This was quite common, in the days before liver was used, during the terminal stage of pernicious anemia A nonbacterial type of pericarditis has been recognized as a frequent complication of lupus erythematosus disseminatus, a condition which may simulate rheumatic fever very closely Hydropericardium is also a common complication of advanced myxedema Finally fluid not infrequently collects in the pericardial sac in cases of generalized anasarca from circulatory failure Here it is a part of the process of edema which produces ascites, hydrothorax and hydropericardium When fluid accumulates in the pericardium with heart failure or in advanced anemic states, it is really not due to an inflammation of the pericardium, but is rather a serous transudate Its diagnosis and treatment will be taken up under the discussion of pericardial effusion

PERICARDIAL EFFUSION

Fluid may accumulate in the pericardial sac in the form of an exudate resulting from inflammation or as a transudate resulting from alteration in the circulation of the blood The latter type can properly be called hydropericardium The diagnostic criteria for the presence of fluid in the pericardium are essentially the same no matter what type of fluid is involved Accumulation of less than 200 or 300 cc in an adult produces such minor changes that it cannot be detected clinically The only condition in which it would be important to recognize such a small amount of fluid is when there is empyema of the pericardium such as may occur in pneu-

monia Here if there is some reason to suspect its presence, the fluid should be sought for by an exploratory puncture

As the amount of fluid increases, certain physical signs may develop The area of cardiac dullness enlarges The change in the outline of the heart from day to day is important, for the actual increase in the dimensions is more significant than the fact that at any one time there is enlargement of the cardiac area A physician who observes the patient from day to day should, therefore, keep accurate data as to the position of the borders of the heart Dullness on percussion will extend outward to the left, although the apex impulse may remain in the same position and, in fact, move gradually inward The presence of appreciable dullness to the left of the impulse is a valuable sign More important still is increasing dullness in the left upper region of the heart This normally is made out at the third interspace or third rib, but in pericardial effusion it may extend as high as the second interspace or even higher Submanubrial dullness also increases, and the right border of the heart extends outward so that with large effusions it may reach the right nipple line Some years ago, it was thought that an obtuse cardiohepatic angle was a very valuable sign of pericardial effusion It has been proved by experiments on cadavers and by careful x ray examination of living patients that this is not generally true I have withdrawn as much as 900 cc of fluid from the pericardial cavity of patients in whom an x ray had shown that the cardiohepatic angle was still acute The general contour of the heart as a result of the effusion becomes more globular and the base of the heart which is made up of the aorta and the great vessels becomes less elongated, and more rounded to conform with the general spherical configuration of the entire shadow As has been mentioned, in rheumatic cases there may be evidence of atelectasis of the left lower lobe of the lung The left lobe of the liver is thought by some to descend as a result of pericardial effusion I have not found this sign of much practical value, for even if the liver is palpable it may be the result of hepatic engorgement and cardiac failure rather than as a specific downward displacement produced by the pericardial effusion

On auscultation, the heart sounds may be found to be muffled and diminished in intensity It is often thought that if fluid accumulates in the pericardium, the pericardial friction sound disappears This is by no means necessarily true, for frequently large effusions will be seen with the persistenct of a loud to and fro rub It has been quite definitely shown that, in some cases of pericardial effusion, there is a true pulsus paradoxus Here the pulse, which is already small, may diminish markedly or actually disappear with inspiration Pulsus paradoxus has been observed in cases of pericardial effusion and found to disappear with the removal of the fluid, only to return when the fluid reaccumulated An interesting observation in this regard was made some years ago showing that most normal individuals mav make the pulse disappear in the radial artery by throwing the clavicle backward entirely independently of the respiratory cycle By this manipulation, the subclavian artery is compressed, obliterating the pulse wave to the arm With normal inspiration, the clavicle is thrown backward

to some extent This mechanism is probably in a measure responsible for the production of pulsus paradoxus The other clinical findings that a patient with pericardial effusion may present are those that are more intimately associated with the underlying disease such as rheumatism, pneumonia, leukemia and the like

X-ray examination serves as a most valuable aid in this diagnosis In general, it confirms more accurately the heart outlines, and gives a better picture of the peculiar globular shape taken on by the heart shadow A further point that is obtained in this way is the lack of demarcation between the left auricle and left ventricle which ordinarily is made out either fluoroscopically or by flat x ray plates On fluoroscopic examination, the individual contractions of the various chambers of the heart are difficult to distinguish, and instead indistinct wavelike movements are seen at the outer portions of the heart The configuration of the heart will also change materially if two plates are taken, one with the patient recumbent and one upright The shadow at the base of the heart will be found broader in the former than in the latter Angiocardiography, involving the injection of opaque medium to visualize the cavities of the heart and the great vessels, can elicit very definite evidence of pericardial fluid There will be a large area between the opaque medium within the ventricles and the outside border of the pericardial sac Although x ray examination is extremely helpful, it is by no means infallible The differential diagnosis generally consists in distinguishing a pericardial effusion from a grossly enlarged or dilated heart This is occasionally quite difficult and at times the only proof will come by making an exploratory puncture of the sac

Pericardial Tapping

The question now comes up when and where should we perform a pericardial tapping? The vast majority of cases with rheumatic pericardial effusions should not be tapped since they so frequently do well if left alone If there were no risk in tapping the pericardium and it were as simple a procedure as a puncture of the pleura it probably would be helpful in shortening the illness of many patients with rheumatic pericardial effusion but there is danger of an immediate fatality The danger does not consist merely of piercing the heart In the experimental animal heart s blood may be obtained repeatedly by puncture without any deleterious effects Another danger lies in piercing one of the coronary vessels These lie very superficially just below the visceral pericardium and have no supporting tissue covering them If one of these vessels is punctured there may be a continuous gradual ooze with a fatal hemorrhage into the pericardial sac I have seen two instances in which fatalities occurred There are times, however, where tapping a rheumatic patient is indicated and may possibly be life saving If the general condition grows worse, the stage may be reached when tapping seems safer than allowing the fluid to remain Exactly how to estimate this point is difficult to describe, and will depend more upon judgment and general experience than upon any single indication If the systolic blood pressure is still maintained satisfactorily it is

generally safe to delay It is surprising how extremely sick such a patient may be and recover satisfactorily without tapping

There are various sites for performing a pericardial puncture Different physicians prefer different procedures and I judge that it is a matter of custom rather than that one method has any advantage over another The method I prefer is to go in at the fifth left interspace, just inside the border of dullness and outside the apex impulse It is well to cocainize the skin at this point The needle is inserted inward, backward and slightly upward One should select a trocar with a dull point to avoid unnecessary scratching and piercing of blood vessels It should be pushed in slowly and it is better to have it attached fairly intimately to a syringe rather than to a suction bottle, for it is then more easily manipulated, and by frequent trials with the plunger of the syringe, one can more quickly and instantaneously tell when fluid is obtained If the needle is pushed in very gradually, applying frequent suction, fluid may be obtained even without ever feeling the pulsations of the heart If these are felt with the tip of the needle, it should immediately be slightly withdrawn and suction continued When fluid is obtained, it should be removed slowly and as much should be taken out as comes freely It is not wise to spend time looking for fluid if none is obtained at the outset, because it is this aimless exploration which may prove disastrous The danger in the puncture is greater if it turns out that there is no pericardial effusion than if a large amount of fluid is present

Another method for pericardial puncture is the so-called "Marfan s procedure This consists of inserting the needle just below the lower border of the ensiform cartilage, going upward, backward and inward This avoids the peritoneal cavity and enters the pericardium from above the diaphragm Another method is to reach the pericardial sac from the right of the sternum, inserting the needle in the fourth interspace about 1 inch from the right sternal border Finally, some clinicians prefer to puncture the pericardium from behind and insert the exploratory trocar below the angle of the left scapula On one occasion I removed 500 cc of fluid from the pericardium by this last method after no fluid had been obtained by exploring through the fifth left interspace In general, it may be said that tapping the pericardium should be regarded as a major procedure and should not be undertaken except after the most careful consideration

In cases of rheumatic effusion, it is practically never necessary to tap the pericardium more than once On occasions, however, within a day or two after the tapping, extensive effusions will develop in the left pleural cavity It is not unlikely that when this occurs, the fluid actually continues to be poured out from the pericardium through the hole made by the trocar into the pleural space I have seen two instances where this occurred, and during this time the patient seemed temporarily a good deal worse than before the pericardium was tapped In each instance, marked improvement resulted after tapping the pleural cavity and removing about 2 liters of fluid

Tuberculous pericarditis with effusion frequently requires repeated tappings in contradistinction to rheumatic pericarditis Favorable results have

been reported from the reinjection of air after the removal of the fluid It has been recommended that when this is done, the volume of air injected should be about half the volume of fluid removed A more promising method of treatment would be the use of streptomycin previously discussed In hydropericardium occurring in cachectic conditions or with advanced cardiac failure, there is little to be gained by tapping the pericardium It is conceivable that under certain circumstances, the patient s life may be slightly prolonged by releasing the pressure on the heart produced by a tense pericardial sac, but unless there is hope that the underlying condition may be improved subsequently, the relief will be only very brief The finding of bloody fluid in the pericardium, not produced by trauma, should make one think of a neoplasm of the pericardium

CHRONIC CONSTRICTIVE PERICARDITIS

The type of chronic pericarditis now to be discussed, i e , constrictive pericarditis is of particular importance because it is amenable to surgical treatment As a result of some previous infection or inflammatory reaction in the pericardium a slow progressive fibrosis results, often with considerable calcification, that produces a constricting influence on the movements of the heart In the course of months or years the heart may become greatly embarrassed so that it is unable to expand or dilate properly It is obvious that if the right side of the heart is prevented from performing an adequate diastole or dilatation, it cannot receive the normal amount of blood It follows, therefore, that the heart cannot expel an adequate amount of blood in any given period of time, for it cannot propel more than it receives The result is a decreased cardiac minute output Because of the impediment to a free inflow into the right heart the venous pressure or back pressure increases This affects the superior and inferior venae cavae as they both empty into the right auricle The pressure in the pulmonary vessels may not be affected unless the restriction in the motions of the left auricle and ventricle is greater than that of the right, which is not usually the case The disturbances in the dynamics of the circulation just mentioned take place irrespective of whether the ventricles are capable of normal systolic contractions or not Even if there were no other abnormalities, such as myocardial valvular or hypertensive disease, and the heart were otherwise perfectly normal, pericardial constriction would necessarily produce these changes There often is another disability resulting from these pericardial scars that makes it more difficult for the heart to contract after it has dilated The pericardium may be bound down to neighboring firm structures such as the ribs, and the work of the heart thereby increased This handicap in contraction is much less important in cases of constrictive pericarditis than is the difficulty in expansion

The etiologic factor in this condition is not always discernible A large number of cases are due to tuberculosis, some to a staphylococcus and others to a pneumococcus infection Other undetermined infections, originally minor in nature, may prove to be the cause of some of the cases that at present have not been classified It is quite evident that in many cases

no definite past history of acute pericarditis can be elicited If the pneumococcus and streptococcus have been playing a frequent etiologic role in the past, realizing that such types of acute pericarditis formerly were very fatal, it may be predicted that with current antibiotic therapy, in many more acute cases recovery will take place and possibly form the background for a larger number of chronic cases of pericarditis in future years A most impressive fact in considering etiology is that rheumatic fever, such a common cause of acute pericarditis, is practically never the cause of chronic constrictive pericarditis However, I have seen one patient with constrictive pericarditis who was considerably improved by surgery for about ten years, who showed definite rheumatic mitral stenosis at autopsy

Symptoms

Let us now consider the symptoms and clinical findings in constrictive pericarditis Such patients may complain of weakness, shortness of breath, abdominal distress and, later, swelling of abdomen and legs The amount of embarrassment that will be present and the severity of symptoms will obviously depend on the degree of constriction around the heart and the stage at which the patient is being observed In general there are two types of conditions that it might imitate and with which it is often confused Because of breathlessness, fatigue and cardiac findings, it may resemble the other more common forms of heart disease and because of the enlarged liver and possibly ascites it may erroneously be regarded as cirrhosis of the liver

Diagnosis

Physical examination of the heart often reveals nothing abnormal The rhythm is generally regular, although in a small number of patients auricular fibrillation may be detected Gallop rhythm is not uncommon No murmurs are present or only a slight systolic murmur may be heard Occasionally a faint, prolonged third heart sound is audible at the apex resembling a faint murmur of mitral stenosis The absence of other evidence of mitral stenosis, such as a dilated left auricle and an accentuated first heart sound, should enable one to distinguish the two conditions The heart is quiet and inactive The customary apex impulse is absent or feeble One of the most significant findings is the absence of any marked cardiac enlargement In almost all other forms of congestive heart failure (with which this is confused) the heart is enlarged This is best determined by x ray examination, which is discussed below The blood pressure is almost always within normal limits or lower than normal and the pulse pressure tends to be decreased The pulse is small, somewhat rapid, and frequently shows a pulsus paradoxus The latter finding consists of a marked diminution or disappearance of the pulse with inspiration, and can be elicited by palpation of the radial artery, or better still, while ausculting during blood pressure determination

The liver is always enlarged and generally palpable, and in later stages ascites may develop In cases of long standing, repeated abdominal taps

may have been performed For a considerable time peripheral edema is likely to be absent but eventually swelling of the legs will appear Probably, as a result of the prolonged hepatic congestion, hypoproteinemia may be present and this in turn helps to produce a stubborn form of edema Unlike other types of heart disease, constrictive pericarditis is characterized by breathlessness only late in the course of the disease and paroxysmal dyspnea or orthopnea does not occur The pulmonary findings may, therefore, be inconspicuous, unless evidence of pulmonary tuberculosis is present Eventually, however, true congestion of the lungs and even hydrothorax may appear The electrocardiograms may be of low amplitude and often show flattening or inversion of the T waves in Leads I or II

The venous pressure is increased This is detected by noting the distention of the cervical veins while the patient is sitting upright or in the semirecumbent position, when normally the veins ought to be collapsed, or in finding distended veins on the dorsum of the hand or forehead Venous pressure in the arms should be determined and will always be found elevated, generally over 200 mm This increase in venous pressure will also be found in any case of right sided heart failure, no matter what the cause may be, and especially in tricuspid insufficiency or stenosis The striking point in constrictive pericarditis is that the venous pressure is elevated when the other evidences of heart failure are not very impressive, it remains elevated even after vigorous methods are employed that are ordinarily helpful in cases of heart disease Finally the pressure both in the veins of the arms and legs is elevated, whereas in cases of cirrhosis of the liver with ascites or in some other abdominal conditions the increase in pressure will be confined to the veins of the legs

An interesting alteration in the dynamics of the circulation is decrease in the minute output of blood What is even more significant is that whereas a normal heart can increase its output per minute five or tenfold with physical effort here the mechanical handicap prevents any such response This explains the fact that these patients may be comfortable at rest but become markedly fatigued on effort or actually find it impossible to do very much physically

The x ray findings are often of primary importance in the diagnosis of constrictive pericarditis On fluoroscopic examination the heart beat is found to be quiet The movements of the cardiac borders are diminished This decrease in contraction of the cardiac chambers, although visible to some extent throughout the heart, is often more marked over certain areas Careful search should be made particularly in the region of the right auricle and ventricle for this is the favorite site for fibrotic plaques to produce constriction Kymograms may furnish permanent records of the actual amplitude of contraction of the cardiac chambers and serve for comparison in judging postoperative results Furthermore in many instances, calcification of the pericardium even to an amazing degree may be detected In some cases, especially those due to tuberculosis, calcification of the pleura may also be found It was previously stated that the heart is not enlarged That is certainly the general rule, but occasionally because the pericardium

is so thick, or because of pockets of fluid, the cardiac silhouette may be increased Although the x-ray findings are very valuable in detecting disease of the pericardium, whether or not the condition causes constriction of the heart must depend on other observations, especially the increased venous pressure, for there are instances of marked pericardial calcification in which the dynamics of the heart are not disturbed

Once the possibility of constrictive pericarditis has been considered, the foregoing features and examinations enable the physician to make the diagnosis These cases are often not detected because the condition is not thought of and will be confused with two general groups of other cases It is among patients who are regarded as having heart failure, in whom the ordinary causes are lacking, i e, hypertension, coronary sclerosis, valvular disease, hyperthyroidism, etc or who are thought to have cirrhosis of the liver, that constrictive pericarditis will be found

Another aid in diagnosis is the response to medical treatment Whereas most cardiac patients with congestive failure improve on rest, digitalis and diuretics, patients with constrictive pericarditis only occasionally show any appreciable response The venous pressure may fall slightly but will remain distinctly above normal despite all efforts In this respect the condition resembles tricuspid stenosis, from which it can be distinguished by the absence of any valvular disease or marked cardiac dilatation

One may repeat that constrictive pericarditis needs to be sought for among patients who, generally 20 to 45 years of age, appear to have some form of heart disease or cirrhosis of the liver, who have a small, slightly rapid, often paradoxical pulse, generally regular in rhythm, although occasionally grossly irregular, who have no hypertension, significant murmurs or cardiac enlargement, and who always show an increased venous pressure and an enlarged liver

Treatment

The treatment for patients with this condition is surgical Some temporary improvement may result from medical management, mainly from diuretics, restriction of salt and fluid intake Digitalis is rarely of value and may aggravate the condition When the protein content of the blood is low a high protein diet should be given and infusions of plasma may be helpful Inasmuch as the fundamental problem is mechanical, surgical resection of the pericardium (Delorme s operation) is the only satisfactory means of relief The surgeon should be one especially expert in cardiac surgery for the operation is difficult and not without risk Owing to the work of White and Churchill, Burwell and Blalock, Cutler and Beck, and others, sufficient progress has been made so that operative results are now very satisfactory Some patients are actually cured and many others are considerably relieved A few are no better because the involvement is too extensive and the surgeon is unable to free enough of the heart to permit adequate expansion Rarely the lung is similarly bound down to the thorax so that respiratory failure continues despite a satisfactory restoration of cardiac mobility Occasionally secondary operations are necessary because

sufficient relief does not result following the first attempt Finally, the physician should be patient in appraising the operative result Although some subjects may make a dramatic recovery in a few weeks, others show little gain for many weeks or even months and yet finally make satisfactory recovery

In performing the operation due regard must be paid to the eventual dynamic relations of the circulation If the right side of the heart is freed of constrictions and enabled to fill normally and the left ventricle remains bound down, the situation may be no better or even become worse than it was before Now, added pulmonary congestion may develop because of the inability of the left ventricle to receive or expel its quota of blood In other words the dissection must be carried out so that a balanced state of the circulation results In fact it would seem wise to free the left side before the right so that even during the operation acute pulmonary edema might be prevented One need have no fear of the ability of the heart to take care of the increase in work, since, in cases of constrictive pericarditis under discussion, the heart muscle can be regarded as essentially normal Finally, it is of interest to know that when improvement or cure is obtained it is maintained Follow up studies so far have shown that constriction does not redevelop, at least for the years that these patients have been observed

CHRONIC NONCONSTRICTIVE PERICARDITIS
(ADHESIVE PERICARDITIS MEDIASTINOPERICARDITIS)

The details of the development of chronic nonconstrictive pericarditis are meagerly understood It is fair to assume that the condition is the result of a single or of repeated infection of the pericardium What is not known is whether recovery in any individual case of acute pericarditis takes place with or without subsequent adhesions One physician may see a patient with acute pericarditis and twenty five years later another physician sees the same patient with heart failure At this time if chronic pericarditis is present it rarely is discovered or if it is correctly diagnosed the past history of acute pericarditis is not known We have very little reliable data from which to ascertain the frequency with which any one of the primary causes of acute pericarditis produces chronic pericarditis The complexity of the problem is increased by difficulties in diagnosis There is reason to suspect that while infection with the tubercle bacillus the pneumococcus staphylococcus, and possibly the streptococcus are responsible for chronic constrictive pericarditis (discussed in the preceding pages) these same infections may account for other cases of chronic pericarditis that are nonconstricting However, when there is also chronic valvular disease with chronic pericarditis it would appear that rheumatic fever is the main etiologic agent Furthermore, it is of considerable interest that such rheumatic cases never produce the picture of constrictive pericarditis

From a clinical point of view cases of chronic nonconstrictive pericarditis may be divided into two groups in one the subjects are symptomless and in the other there is significant heart disease and eventually heart failure

serositis On postmortem examination the pericardium was found to be perfectly normal, the heart was enormously enlarged and dilated, and there was stenosis of the mitral, aortic and tricuspid valves This well illustrates the difficulties in diagnosis

Treatment

As has been indicated, the importance of accurate diagnosis of chronic pericarditis of the constricting type is great because surgery offers the only effective treatment This is not true of chronic nonconstrictive pericarditis or chronic adhesive pericarditis For the most part, this condition occurs in patients having rheumatic valvular disease and enlarged hearts They suffer from cardiac disabilities that are not particularly the result of the adhesions Such patients would be receiving treatment for heart failure anyway and in most cases the treatment would be no different even if it were known that adhesive pericarditis were present

Many years ago Brauer devised an operation for the relief of chronic pericarditis which he called cardiolysis' At that time the essential differences between the constricting and nonconstricting aspects of pericarditis were not so well differentiated as they are today The procedure consisted of removing several ribs and a portion of the sternum over the precordium, thus permitting the heart to tug away at flexible soft tissue rather than at an unyielding bony chest This operation did not necessitate the exploration of the pericardium and heart Although carrying much less risk than the Delorme operation, in which the heart itself needs to be explored, it is performed very little nowadays It can do no good for constrictions that may be present Its only effect would be to diminish the work of the heart in its contraction or to give an enlarged heart a little more room, serving as a decompression of the chest Inasmuch as the pericardium is not explored, the presence of pericardial adhesions is not confirmed by the operation Some patients appear to have been improved by this operation and in a few of these who were subsequently examined post mortem no pericardial adhesions were found Improvement could then only be ascribed to a decompression effect on an enlarged heart

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Angina Pectoris and Coronary Thrombosis

ANGINA PECTORIS

Angina pectoris is a distinct clinical entity in the sense that when properly understood it can be diagnosed accurately, the subsequent course of the disease can in a great measure be predicted and the type of pathologic changes in the heart at autopsy can be foretold. The likelihood of sudden death is a necessary corollary of the term 'angina pectoris.' In addition, one may expect certain measures that are helpful in some heart conditions to be comparatively useless in angina pectoris and other means not ordinarily employed to be of benefit. For example, nitroglycerin may relieve an attack of angina and have no influence on the precordial pain of cardiac neurosis or mitral stenosis. It, therefore, becomes imperative to isolate from among those patients who suffer from chest distress the ones who have true angina pectoris.

A patient either has or has not angina pectoris. There is no room in this discussion for terms such as pseudo-angina, false angina, juvenile angina and anginoid. The use of such terms has served to spread confusion and to distort the truth. It may be proper to call a condition pseudo angina if the symptomatology is quite characteristic of angina and yet one infers that there is no true anginal element whatever in the problem. Furthermore the term 'mild angina' is often equivocal. If the terms mean that the complaint is not very troublesome its use is proper, but it must not be inferred necessarily that the disease is mild or trivial. An anginal patient with only mild complaints may be dead of heart disease in a few days, for the underlying process may yet be grave.

Much of the confusion concerning angina has resulted because of the lack of uniformity in the explanations of the mechanism of the attacks that characterize the disease and in the underlying etiology. So divergent have the hypotheses been that one authority has insisted that angina has nothing to do with the heart and is only the result of disease of the aorta, another has maintained that it is due to spasm of the coronary arteries, a third that it is due to heart fatigue and a fourth that it is a neurosis. Although it is not proposed here to discuss the various theories concerning angina pectoris, one thesis propounded seems to be worthy of mention. It has been suggested that the invariable mechanism which causes an attack of angina is

anoxemia of the myocardium no matter how brought about This can explain the overwhelming frequency of angina pectoris in disease of the coronary arteries It also accounts for its occurrence in some cases of anemia with comparatively normal coronary arteries, for here anoxemia of the heart might well result from lack of hemoglobin It also accounts for the disappearance of angina in some cases of thyrotoxicosis when the demands on the heart are decreased by diminishing the body metabolism There are many other features of angina that this hypothesis satisfactorily explains which other theories do not and I feel that at present it is the one most worthy of our consideration

Etiologic Factors

Although there is no single specific organic cause of angina pectoris there are numerous etiologic factors which are definitely related to its development It has generally been known that it occurs in males more frequently than in females, the proportion being about three or four to one This disproportion becomes all the more impressive when it is appreciated that hypertension is more frequent in females than in males It at least throws some doubt on the significance of hypertension per se as an etiologic factor Probably the most important etiologic factor is heredity There is a strong familial factor in angina pectoris All practitioners who have treated members of the same family over long periods of years have noted that there are families with a tendency to sick headaches, others that are neurotic, some with a tendency to asthma and still others with a liability to early death from one or another form of cardiovascular disease I have seen three brothers all of whom died of acute coronary thrombosis during the sixth decade of their lives It cannot be mere chance that three members of the same family should die of the same disease at approximately the same time of life It seems as if the original tissue or structure that such individuals have at birth must be peculiar, so that ordinary wear and tear does more harm to certain vessels than is the case in other people It is not altogether too fanciful to conceive that the anatomic configuration of one of the coronary arteries may be slightly different, so that with the repeating contractions of the heart muscle one particular part of the vessels receives undue strain or tension Even a very slightly exaggerated bend in the course of a vessel could easily account for its premature degeneration This peculiarity would readily be inherited just as are queer configurations of the lobes of the ears and other structures It would also explain the decided susceptibility to occlusion of one special portion of the left descending coronary artery, about 2 cm from its origin In support of the idea that the inherited type of the coronary arteries may be of importance is the recent work of Dock He has found that in the male sex, from infancy on, there are more extension areas with thicker layers of endothelial lining than in the female This is the first light to be thrown on the sex discrepancy in angina May it not be that those who are born with a greater degree of thick endothelium develop more atheromatous changes in these areas? Another possible explanation of this familial factor of angina pectoris and its pre-

dominance in the male sex is that the peculiarities in the structure and function of the endocrine system which we inherit may be the basis of premature arteriosclerosis

As a corollary of the hereditary influence there is a distinct constitutional type of individual who is particularly susceptible to angina pectoris I refer to the well set, strong man who is slightly overweight and who has been especially healthy all of his life So often these patients boast of their previous good health and remark that they never had a sick day in thirty years,' and that they "could do the work of two men They have been physically stronger than the average, even when their daily activities did not require them to use that strength The forearms of these individuals are muscular, round and the skin fits snugly over their muscles in contrast to the flat-armed type It is very striking that angina is quite rare in the undernourished and in those who have been sickly for years It selects the vigorous rather than the weak From an anthropologic point of view it is most common in the mesomorph (muscular), less common in the endomorph (fat) and least common in the ectomorph (thin)

The average age of patients with angina pectoris when the disease first manifests itself is about 56 for males and about 58 for females There are a few young individuals even in the second and third decades with angina who suffer from aortic valvular disease This relation will be discussed in great detail later Even apart from these exceptional instances, angina is not extremely rare in the fourth decade and I have even seen an instance of typical angina pectoris followed by acute coronary thrombosis in a young man 24 years old, and one in a woman of 22 who subsequently went through pregnancy successfully It seems to be occurring in younger individuals than was observed a few decades ago

When conditions such as Buerger s disease hyperthyroidism and aortic valvular disease, to be discussed below are not present, young anginal patients between the ages of 20 and 40 will generally be found to have other etiologic stigmata, such as a marked family history of vascular vulnerability, diabetes or hypercholesteremia

It is the opinion of most observers that angina is increasing in prevalence both relatively and absolutely It is obvious that with the marked increase in the span of life that has taken place during the past century and with the improvement in the methods of diagnosis, there should be a greater number of cases of those diseases that mature in persons in the second half of life However, there is indirect evidence to show that angina is actually more common among a given number of adults than formerly I am of the opinion that its relative and total incidence has been on the increase and that this increase is real rather than apparent The reason for this is very obscure It is generally explained on the basis of the stress and strain of modern life This still leaves us in a quandary as to the actual mechanism involved Has there been some specific deleterious influence at work during these past decades such as the telephone with its terrorizing clang, or the motor car with its noxious fumes or a dietary defect with an overabundance or lack of some element, or the prevalent habit of smoking? Is it even due to

a process of natural selection, those with angina being the more fit and dynamic and propagating their kind? or is it merely the hustle and bustle of the twentieth century? These are important but unanswered questions

In a very recent study it was found that Jewish patients with coronary artery disease died on the average at a considerably younger age than non-Jewish patients A similar though less striking difference was found in the age at death of smokers in contrast to nonsmokers, the former dying about three years earlier If this can be confirmed and it can be shown that the overall age of the groups studied were the same as the average age of all patients with angina, it would follow that these two factors are somehow related to the development of coronary disease

It is of some interest that females are not only less frequently afflicted with angina but that the disease occurs on the average two to three years later than it does in the males The reason for this is obscure but the facts are beyond dispute This may be one of the important reasons that the average age of women at death in the latter decades of life as shown by all insurance statistics is consistently three years or more greater than that of men If the smoking ot tobacco has had something to do with this difference in the sexes, as has been thought by some, it can be expected that in the next few decades, as a result of the greater prevalence of smoking among women, the discrepancy beyween the sexes may disappear or diminish in extent Arteriosclerosis is much less extensive in the female than in the male sex This merely puts the problem in different words, for the reason for this lessened susceptibility to sclerosis remains obscure

Among the specific di eases that are etiologically related to the develop ment of angina pectoris *syphilis* used to be regarded as most important It was particularly looked upon as a frequent cause of angina in young people Since the introduction of the Wassermann reaction it has become evident that syphilis is an uncommon finding in angina I found it present in only 4 to 5 per cent of the cases and even in these it cannot always be regarded as directly related to the angina There has been considerable confusion between syphilitic aortitis and angina pectoris The two conditions are quite different One need but realize that the former is extremely common in the colored race whereas the latter is very rare, to question any important bearing that syphilis could have in the development of angina There are occasional instances in which the syphilitic process extends down the ascending aorta and partially or completely occludes the openings of the coronary arteries In this way anginal distress may result Furthermore, the development of aortic insufficiency itself with its accompanying low diastolic pressure has been regarded as a cause of angina I have considerable doubt as to this relationship However, when there is involvement of neither the coronary orifices nor the aortic valves, it is very unlikely that syphilis is responsible for angina, even when both syphilis and angina are present In a similar way a small number of tuberculous individuals may incidentally have syphilis without any relationship between the two con ditions It is important to bear in mind that syphilis very rarely produces changes in the main course of the coronary arteries where the customary

atheromata are so common and that when a syphilitic has such changes it does not follow that the coronary changes are syphilitic or in any way different from those seen in nonsyphilitics

A much more important disease related to angina is *diabetes* A large number of diabetics, especially the elderly mild ones, often eventually develop coronary artery disease This relationship probably will grow more common as time goes on, for insulin should prevent early death in diabetes and permit such individuals to live long enough to develop the vascular changes that are so prevalent in this disease It is not altogether certain that diabetes is the actual cause of such angina pectoris as develops for the age at death of patients with angina pectoris and diabetes is essentially the same as those without diabetes It may be that diabetes merely indicates the type of individual who has a vascular vulnerability just as the family history and constitutional type reflect those prone to this disease For the most part it is the mild diabetic 50 or 60 years old, who has not required the use of insulin and in whom the diabetes may be looked upon as one of the evidences of generalized arteriosclerosis who develops anginal symptoms

A high cholesterol content in the blood (*hypercholesteremia*) is common in patients with coronary artery disease This is particularly true of those who have xanthomatosis or even when only isolated xanthelasma lesions are present around the eyelids It has been maintained by Leary that diets high in cholesterol content produce marked sclerosis of the arteries especially the coronary arteries, in the experimental animal Whether this mechanism applies to man and whether it can be altered by diets that are low in animal sterols or by thyroid administration has not been determined

Similarly there are other less common diseases that are particularly associated with generalized arterial changes which are etiologically related to angina pectoris I have reference to *gout, chronic lead poisoning Buerger s disease myxedema Paget s disease* and *polycythemia* In these conditions the development of angina pectoris can readily be visualized as the result of alterations in the coronary arteries similar to changes going on in other vessels of the body The development of angina in association with Buerger s disease is of especial interest because it accounts for some of the cases of angina occurring in younger individuals in the thirties and early forties

The presence of aortic insufficiency or stenosis is attended in some cases by typical attacks of angina This relationship has been explained but not altogether satisfactorily, by the following mechanism The coronary arteries are thought by some investigators to be nourished during the diastolic pause of the heart beat, for when the heart is in systole the contracted muscle forcefully occludes the vessels and prevents any flow of blood During the diastolic relaxation however, the coronary arteries are open and it is then that the heart muscle is nourished Aortic insufficiency is frequently accompanied by a low diastolic pressure which results in a diminished nutrition of the heart muscle It is this relative anoxemia of the heart that is supposed to make it more susceptible to attacks of angina This mechanism does not explain the exact precipitating causes of the attack for the defect of the aortic valve is permanent and constant, present when the patient is

free from attacks Some other factor is required that sets off the spark to produce a spell of anginal pain Other possible explanations that bear on the relationship between angina and aortic valvular disease were taken up under the discussion of aortic stenosis (Chap 4) The relationship of aortic valvular disease and angina explains the occurrence of the latter in a small number of young individuals There are persons of both sexes in the second and third decades of life with rheumatic aortic valvular disease who have typical attacks of angina It is important to understand this clearly because although such individuals are liable to sudden death, a characteristic of all cases of angina, they are apt to live many years The prognosis for duration of life after symptoms develop is generally better than in ordinary cases of angina, because their coronary arteries are essentially normal They do not as a rule develop coronary thrombosis but rather continue the course of valvular disease and eventually die of congestive heart failure or bacterial endocarditis

There remain a few other conditions that need consideration I refer to *severe anemia, hyperthyroidism* and *paroxysmal rapid heart action* If a heart that is otherwise competent and structurally sound is nourished by blood which contains 20 per cent hemoglobin or less as may occur in pernicious anemia, one can readily see that a state of relative anoxemia may exist in the heart muscle The same ischemia could result with normal vessels and impoverished blood as occurs with normal blood and narrowed vessels To be sure, anginal attacks are not common in anemia, probably because there are other compensatory mechanisms that come into play to help nourish the heart and other tissues, such as an increase in the velocity of blood flow and in the minute output of the heart, an increase in the utilization of oxygen and arteriolar and capillary dilation However, such attacks do take place although it is to be expected that they will occur more frequently when the anemia is marked and when it develops in an individual whose coronary arteries are already showing degenerative changes It is obvious in these cases that the successful treatment of the anemia may prove helpful for the attacks of angina and may even make them disappear entirely Most cases of angina occurring with anemia really have a significant degree of coronary sclerosis I have seen one instance, however, in which classical angina was associated with severe refractory aplastic anemia in a young woman 32 years old who showed normal coronary arteries post mortem

This aspect of anemia and angina has been generally emphasized, but an opposite clinical relationship has received very little attention I have seen several instances of pernicious anemia in which cardiovascular accidents developed directly after the red blood cell count returned to normal In one such case, as the red blood count went from two to five million in three months, the systolic blood pressure rose from 140 to 240 with the development of hypertensive headaches and a terminal fatal cerebral hemorrhage Retrospectively, it would have been wiser to diminish the liver therapy and to rest content with a blood count of four million, notwithstanding the slight risk of neurologic complications Similarly, several cases of coronary thrombosis have occurred shortly after the restoration of the

red cell count to normal in primary anemia One, therefore, needs to be on guard for such exceptional complications

For quite a different physiologic reason angina pectoris may result from active hyperthyroidism Here the mechanism is fairly clear, for a heart that is competent when the bodily demands are normal may find itself embarrassed if the basal demands are increased 50 to 60 per cent Such an individual may be compared to one who is forced to walk all day long The heart again is suffering from relative anoxemia because of excessive demands It is unlikely that a normal heart will be so affected by an elevated metabolism What is more probable is that the thyrocardiac patients who have angina pectoris have some disease of the coronary arteries which requires an increased demand on the heart to produce embarrassing symptoms Here also the cure of the hyperthyroidism may eliminate or greatly improve the anginal attacks

Finally there is a form of angina pectoris that can occur in an otherwise healthy heart as a direct result of a sudden increase in its rate Any of the forms of paroxysmal rapid heart action discussed in Chapter 13 may be the direct cause of such attacks Although this relationship is not common, when it does occur it may offer considerable difficulties in interpretation These transient irregularities may result from an attack of true coronary thrombosis and yet they can occur in an otherwise healthy heart and resemble acute coronary thrombosis I saw a man thirty-five years ago who had severe anginal attacks during bouts of paroxysmal auricular tachycardia when his heart rate would reach 250 and be maintained there for hours and days He never showed any evidence of organic heart disease remained in normal health for over twenty years and only in later years has there appeared angina on effort not associated with tachycardia The probable explanation of the anginal pain under these circumstances is that while the heart rate is extremely rapid the pulse pressure becomes so low that there is very little effective head of pressure to drive the blood around In the case cited above, the blood pressure for hours and days during attacks was about 96 systolic and 88 diastolic The coronary artery flow must have been greatly impeded with a resulting anoxemia of the heart It is evident that such paroxysms of rapid heart action can occur in diseased hearts as well, but that when they occur in normal hearts the prognosis can be excellent, for the arrhythmia is generally within control or at any rate self-limited in its duration

The above peculiar forms of angina pectoris including those with aortic valvular disease form only a small portion of the entire group, possibly 5 to 10 per cent All the others have no associated anemia hyperthyroidism or valvular disease, and represent the type that is commonly met with in general practice One may ask, what is the basic cause of the attacks in these individuals? There have been endless discussions and numerous theories as to the cause of angina and the final solution has not yet been reached The evidence points more and more to the view that anoxemia of cardiac muscle is the most important factor An observation which supports this theory is the reproduction of anginal attacks in patients suffering from this

disease by the inhalation of air containing decreased amounts of oxygen The theory of aortic origin of anginal pain is losing ground and now most authorities look to the coronary arteries for the underlying defect I have never yet failed to find some disease of the coronary arteries in any of my own patients with angina that have come to postmortem examination, with the exception of the few who had some other condition, such as valvular disease or anemia It is conceivable that under most unusual circumstances even fatal angina might occur with a structurally normal heart Men working with nitroglycerin have been known to drop dead instantly on slight effort after being away from their work for a few days These observations have been made by Cecil K Drinker and lend support to the theory that the mechanism of coronary spasm is a cause of angina

To be sure, there are patients who pathologically show sclerosis of the coronary arteries in whom angina was not detected in life This, I believe, does not invalidate the coronary hypothesis as the origin of angina Even if the coronary view is accepted it is not clear whether the pain is the result of spasm of the coronary vessels, whether it is due to the dilation of the vessel proximal to the spasm or whether the pain sensation comes from metabolites that are not adequately removed from the area of relative ischemia of the heart Finally, a most important unsolved question is the precipitating cause of an attack which occurs while the patient is at rest Organic changes in the heart do not come and go The coronary artery is no less sclerosed a few minutes before than during a typical attack of angina What trigger mechanism lies behind the temporary explosion? May it not be the endocrine system, particularly the adrenals or thyroid gland? Some experiments I performed several years ago suggest this hypothesis for it was found that adrenalin almost invariably brought on typical attacks in patients suffering from angina and had no such effects on control individuals The whole problem of anginal pain, although nearing a solution, still has many mysteries to be cleared

Diagnosis

The diagnosis of angina pectoris depends on the proper interpretation of symptoms For this reason the history is all important The patient will complain of a peculiar distress in the chest It is frequently not described as a pain but rather as a disagreeable feeling like 'indigestion" It is so frequently associated with the belching of gas that both the patient and the physician are only too prone to regard the condition as abdominal in origin It is well for the physician to elicit carefully the character and location of this complaint Too often he regards the complaint as a pain in the precordium when it is neither a pain nor located over the heart Most commonly the location is in the center of the chest, in the sternum or just to the left of the sternum Much less frequently it is located over the precordium and very rarely at the apex of the heart It may arise anywhere from the epigastrium up to the root of the neck and characteristically radiates down the arms, through to the back, the neck, the jaw, the teeth, the face or to the shoulder The radiation is more common to the left than to

the right, but not infrequently both sides are involved Occasionally the pain starts peripherally and radiates to the chest and rarely it is only in the arms or back and not in the chest The discomfort is generally described as a constriction, a feeling of tightness, a burning sensation, a feeling of fulness, a choking, a distress or just an uncomfortable pain Some will say that their clothes suddenly feel tight and others will state that they want to lift or loosen their shirt The peculiar terms used by different patients to describe the symptoms are very illuminating and it is well to write down the history in the very terms used by the patient In this way the physician quickly learns the character of the pain and becomes more proficient in recognizing this peculiar type of distress Often the complaint is so mild that the patient refuses to call it a pain and resents taking it very seriously The mild complaint may nevertheless be very ominous

The manner of development of the distress is also peculiar It comes fairly suddenly and especially on effort The activity that is most likely to bring on a spell is walking Patients often are able to do a great deal of hard physical work indoors and yet not be able to walk a block or two in the street The effort of walking up a slight grade is particularly difficult There are two other factors that accentuate this distress These are a full meal and cold weather Frequently patients may be able to walk to their luncheon but have difficulty in returning, or may have attacks when the air is cold or the wind blowing and none when the weather is warm Chilling only a part of the body by placing ice cubes in the hands can diminish the exercise tolerance of patients with angina so that attacks come more readily Another peculiarity is that the pain may come at one time after a brief walk and than at other times not recur after a much longer and more difficult walk Some will complain that they may have an attack during the first or second hole of golf and then no more during the rest of the game, or have a spell going to the train after breakfast and be free the rest of the day Furthermore there are peculiar efforts in individual cases that bring on attacks such as shaving in the morning, taking a bath, undressing at night or getting into bed between cold sheets Only careful history-taking will uncover these variations in symptomatology

Next in importance to physical effort is mental excitement or agitation There are many patients who will have an attack on coming into a consultant s office In this way I have frequently been able to witness in my office the entire development of the anginal spell Sexual intercourse is another precipitating factor that is not uncommon A heated argument, suddenly being informed of pleasant or unpleasant news or any form of nervous excitation may be the precipitating cause of attacks Finally anginal attacks may come without any apparent precipitating cause while the subject is at rest They may even awake patients from sleep, in such instances we try with only partial success to blame the attacks on dreams A division has been made by some observers between angina of effort and angina of rest but I see no useful purpose that can be attained by this distinction The latter frequently follows the former although occasionally the first attack may be of the form described as angina at rest There are no import

ant practical differences between the two types except that it has seemed to me that those who have angina and hyperthyroidism are very prone to have attacks at rest

When the attack comes, the patient is generally brought to a sudden halt When it occurs in the street he is apt to walk slowly to a shop window, not to attract attention, and wait for the distress to relax He quickly learns that if he remains stationary for a few minutes the pain disappears and he is able to continue He also learns that if he does not stop the pain persists and may grow more uncomfortable In rare instances the attack does not prevent continuation of the effort During the period of pain in most instances there is no shortness of breath, but some will complain that they have difficulty in breathing It is hard to distinguish true dyspnea from a certain immobility of the chest that may accompany the attack The patient may not want to move his chest even by the act of breathing for fear of aggravating the pain When true breathlessness occurs with the attack it generally signifies that there is an additional factor of heart muscle insufficiency which for the most part is absent in angina The occurrence of fear of death is variable It is often, but by no means always, present It will depend on the severity of the pain and the psyche of the individual There is something fearful and ominous about the sensation, however, even though it is not severe The same patient who dreads an attack of angina may suffer a very violent attack of renal colic requiring repeated hypodermic injections of morphia yet experience no fear

There are occasional instances in which attacks of angina are related to hypoglycemia Patients may observe that spells are most likely to occur when they are hungry, or may be prevented by eating something sweet One should particularly suspect this relationship if attacks occur late at night In these cases blood sugar studies are useful

There are other cases in which attacks have developed directly after severe trauma to the chest or after a very violent sudden effort There is now reason to believe that under such circumstances the coronary arteries may be actually traumatized with resultant symptoms of coronary insufficiency The relationship between trauma and coronary artery disease is a very difficult one to disentangle, because so much depends on subjective complaints which may not be reliable when matters of litigation arise However, when it is known that an individual was well before such an accident and begins to suffer from definite anginal pain hours or a few days afterwards, it is reasonable to assume that there is a relationship of cause and effect The same is true when one suffering from simple angina becomes markedly worse or develops evidence of a coronary thrombosis directly after significant trauma

When a patient is seen during an attack he will appear slightly pale and introspective and there may be slight perspiration He will not want to converse but will prefer to remain quiet until the attack is over The pulse *rate during* this time may be unchanged but more generally is somewhat accelerated The rhythm remains regular in the vast majority of cases but occasionally extrasystoles may suddenly appear during the attack Such

patients may be helped by quinidine The blood pressure, if taken during the pain, will rise almost invariably It is not the pain that produces the rise in pressure in these patients for I have found no rise in these same persons during the pain of severe renal or biliary colic Occasionally there is an increased flow of urine or of saliva during attacks After a few minutes, either spontaneously or as a result of a nitroglycerin pill, the distress disappears and the patient feels quite well again

The preceding details have been gone into because upon their proper evaluation will the diagnosis of angina pectoris depend in the vast majority of cases The physical examination and all our laboratory tests are so often of no help that it is only from a true understanding of the nature and character of the symptoms that these cases will be recognized It is the failure on the part of the doctor to appreciate the importance of mild distressing complaints in the chest that leads many patients with angina pectoris to be overlooked entirely or to be treated for indigestion or gas It is imperative that we deliberately inquire into the history of all our patients over 40 years of age, asking whether they have chest distress on effort no matter what their primary complaints may be, for they may not volunteer this information The lesson that needs to be repeatedly emphasized is that this common and serious malady is generally associated with no significant abnormal findings on physical examination

Physical Examination and Laboratory Tests

Although there is very little to be made out on examination that is characteristic of angina one frequently detects some abnormalities in the cardiovascular apparatus Generally there is evidence of arteriosclerosis in the peripheral vessels or on ophthalmoscopic examination Often, however, there is no more than would be expected at that age and at times there is even complete absence of any detectable vascular disease The blood pressure is variable and may be anywhere from subnormal to markedly elevated

There is a curious and most important difference in the blood pressure findings in the two sexes Whereas the average readings for males were found to be 149 mm systolic and 89 mm diastolic the corresponding figures for females were 190 mm and 102 mm In fact, there were very few instances in a considerable experience in which I found a systolic blood pressure under 140 mm in a female except a few in whom a previous hypertension had given way to a lower pressure after a coronary thrombosis The inference is that although normal or low blood pressure is common among males with angina the absence of hypertension in females, except following a coronary thrombosis, makes one strongly doubt the diagnosis

The heart in most instances is enlarged although frequently the enlargement cannot be detected without x ray examnation Occasionally calcification of the coronary arteries can be seen fluoroscopically or on the x ray film Although such calcification always means coronary sclerosis, is distinctly pathologic and generally establishes the diagnosis of angina, it occasionally may be found in the absence of angina It must be remembered that the term angina pectoris denotes a physiologic state, and sclerosis

of coronary arteries is a structural condition Although the latter is by far the most important cause of the former, each on occasions may exist without the other

There is a dominantly regular rhythm in *practically all cases Occasional* extrasystoles may be present but auricular fibrillation is rare There seems to be some antagonism between auricular fibrillation and angina If the former exists it is unlikely that the latter will subsequently develop The presence of both does occur but is rare Auricular fibrillation can develop in a patient with angina pectoris following an attack of coronary thrombosis or after angina has been present for some time

The heart shows no murmur whatever in about one half of the cases and in the other a systolic murmur is heard at the apex or base of the heart A small number, possibly 5 to 10 per cent, show a diastolic murmur This includes the very rare patient (1 per cent) with mitral stenosis who has angina, the young group of rheumatics with aortic valvular disease and a few older patients with aortic insufficiency due to syphilis or hypertension On the whole the quality of the heart sounds is of no aid in diagnosis In most instances the sounds are normal in character but in some the intensity of the first heart sound is distinctly diminished A diminution of the intensity of both heart sounds can be due to a thick chest wall or overlying emphysematous lungs and the like, but if the first sound is muffled and the second sound is distinct, it at least makes one suspect that the heart muscle is diseased In ome a gallop rhythm may be heard There is no evidence of congestion in the liver, lungs or extremities unless there is an added element of congestive heart failure which will be found in a comparatively small number

There are two other examinations commonly carried out in studying patients with heart disease, i e, determination of the vital capacity of the *lungs and electrocardiography* The *vital capacity is the greatest amount of* air one can expire after inspiring as much as possible This varies mainly with the height of the body Normal standards have been established and whereas formerly they were calculated with reference to given heights this was later changed and comparisons were made with reference to the surface area of the body In this latter calculation the surface area is determined from the height and weight of the body This change in calculation seemed more scientific and accurate when it was first introduced, but I have always felt that the reverse is true One can obtain a closer reading of the expected or normal vital capacity from the height than from the surface area of an individual The following example will make this clear A young man 25 years old, in good health, weighs 135 pounds and is 67 inches in height His vital capacity is 4000 cc, which is perfectly normal In ten years he gains 40 pounds, is still in perfect health, the vital capacity is still 4000 cc and his height is the same The surface area has increased and the expected or normal vital capacity has increased Calculating on the basis of height his vital capacity is 100 per cent of normal, while in terms of surface area it has fallen to about 80 per cent This gives one a false impression, for his circulation has been normal throughout There is no reason why one should

expect a greater vital capacity because the subject has become obese In fact, the reverse occurs when obesity is marked A patient may have no disease of the cardiovascular system and yet have a distinct diminution in the vital capacity of the lungs from obesity alone When the added weight is sufficient, it may inhibit the expansion of the chest and the descent of the diaphragm This often results in dyspnea on effort This type of diminished vital capacity and shortness of breath does not indicate heart disease is not of itself progressive and must be carefully distinguished from the other more serious type of breathlessness This may entirely disappear with appropriate dieting and a consequent loss of weight A corollary that follows from the foregoing is that a thin cardiac who is short of breath is apt to be in a worse state than an obese cardiac, for, in the case of the latter, part of the dyspnea at least is due to the benign obesity Similarly, a low vital capacity reading is more ominous in a thin than in a stout individual It is apparent that the readings must be interpreted in the light of the constitutional type or physique of the individual

Age likewise influences the vital capacity to some extent Practically all the normal standards with which readings are compared have been obtained from young men and women, generally college students or young hospital interns and nurses There are no extensive figures for normal older people The result is that the vital capacity of patients with angina pectoris is being compared to normal young people The readings thus obtained would indicate that in angina the vital capacity is diminished about 20 to 25 per cent If suitable comparisons could be made this difference would be much less and probably would show that when unassociated with myocardial insufficiency the vital capacity in angina is essentially normal What has just been stated about the vital capacity in relation to age and weight needs to be borne in mind in interpreting readings It is difficult to compare the reading in one patient with that of another unless the differences are marked, and yet smaller changes occurring in the same individual from time to time may have more definite significance This is particularly true in following the same patient over the course of years

In the interpretation of the relation of the vital capacity of the lungs to breathlessness, two other factors need consideration The first is the general *muscular strength or tone of the body* especially the muscles of respiration With marked anemia or with general asthenia, although the total breathing space is normal, the effort of taking deep breaths rapidly is exhausting and cannot be kept up for any length of time and breathlessness results The second is the speed with which the respiratory cycle can be completed In marked emphysema, expiration is so difficult and prolonged that the number of respiratory cycles per minute is considerably reduced In the last analysis it is not only the actual vital capacity of the lungs but the facility with which a given amount of air can be inspired per minute that determines the development of dyspnea

The *electrocardiographic findings* in angina pectoris are variable Frequently they are perfectly normal or they show preponderant hypertrophy of the left ventricle which has no diagnostic significance On the other

hand, in a good many cases there will be distinct abnormalities of the ventricular complex Not all of these changes are particularly indicative of angina In fact, some of these are also found in association with heart disease other than angina, but in so far as they direct attention to a pathologic condition of the heart they aid in avoiding the error of making a diagnosis of *normal heart* Although a Q wave in Lead III is found not infrequently in a person with a normal heart it has appeared to be much more common in those suffering from coronary artery disease There are other changes that point more clearly to angina pectoris inasmuch as they are so commonly associated with disease of the coronary arteries Among these are spread and delay in the QRS complex, bundle branch block and initial ventricular complexes of low amplitude All such alterations occur in heart disease other than angina but for the most part are not found in normal hearts Other abnormalities that more clearly point to coronary artery disease are certain changes in the QT complex or in the T wave These changes will be discussed in greater detail under coronary thrombosis but in so far as some of them are observed in cases of angina they often are diagnostic Occasionally opportunities have been afforded for obtaining electrocardiograms of patients during an attack of angina and some have shown slight but distinct alterations of the RT complex resembling those seen in coronary thrombosis The R-T segment is somewhat depressed or elevated in one or another lead as compared to the form that is seen in the tracings before or after the attack This has been regarded as additional evidence to support the theory that anginal attacks are due to myocardial ischemia The importance of electrocardiograph is that, although often it is of no help, in some doubtful cases of angina the tracings may be the only evidence of the grave condition that exists and that without such evidence the patients would be regarded as having a normal heart

When the diagnosis of angina is still in doubt there are some tests that may prove helpful The first is to have the patient exercise with the purpose of bringing on the distress of which he complains This can be done by making him walk upstairs until pain is produced, or by using a so-called "two-step' test One might still be in doubt whether the type of chest pain thereby brought on is anginal or not Another test is to have the patient breathe an atmosphere poor in oxygen content (10 per cent) If this is continued for ten to twenty minutes, anginal attacks may be reproduced in those suffering from this disease Furthermore, breathing low concentrations of oxygen may produce abnormalities in the R-T segment and T wave which do not occur in normal hearts Somewhat similar electrocardiographic changes may be produced by a brief effort When these are marked such as shown in Figure 160 the diagnosis of coronary sclerosis is quite clear However, an appreciable number of cases of angina (approximately 30 per cent) may have a negative anoxia test Finally, I have employed an adrenalin test for angina that merits a word of explanation It was found that the subcutaneous injection of 0 5 cc to 1 cc of 1 1000 solution of adrenalin reproduced attacks of angina pectoris in most cases and failed to do so in control cases In some patients even smaller doses were effective

Unfortunately adrenalin injections are dangerous when given to patients who have heart disease and especially angina This test, therefore, should never be employed when the diagnosis is quite definite and the amount employed in performing the test should be small at first (e g , 0 3 cc), increasing the amount if necessary on subsequent examinations When positive, the patient will start complaining of the same sort of pain in the chest five to fifteen minutes after the injection is given The pain will generally be accompanied by a rise in pulse rate and in blood pressure Just as soon as the pain is reproduced nitroglycerin, amyl nitrite or, if necessary, morphine is given to bring the attack to an end I believe that the test can be helpful in some doubtful cases, particularly when it is felt that the condition is functional rather than organic It must be borne in mind that performing any functional test for angina is dangerous, for instant fatalities have occurred

There are several conditions that particularly must not be confused with angina First, there is the large group of patients suffering from some form of organic heart disease, valvular or hypertensive, who complain of pain in the chest For the most part the pain is at the apex, not in the sternum, and is often associated with hyperesthesia of the left breast Then there is the group who have functional heart disease Here the pain is also apical and generally comes while at rest Furthermore, arthritis of the spine, subdeltoid bursitis and muscular pains from trauma and similar conditions may simulate angina The confusion becomes increased because many patients with coronary disease develop a troublesome pain in the left shoulder, arm and hand that resembles causalgia and is not due to the heart They may, therefore, have two different types of pain in the left arm, one of an anginal type brought on by effort and another related to motion of the left arm or shoulder The two need to be carefully distinguished, because of differences in treatment For the shoulder pain local heat and aspirin may be all that is required occasionally stellate ganglion block will be helpful Quite recently disabilities in the hands have been reported as associated with coronary artery disease, especially following myocardial infarction Dupuytren s contraction and stiffness of the fingers resembling sclerodactylia presumably due to vascular constriction have been observed These various features are distinct enough to be called the shoulder arm syndrome and is a frequent complication in patients suffering from coronary artery disease It is much more common on the left side but may involve the right A most important and common differential diagnosis is that between angina and gallstones The difficulty is increased because so many have both conditions X-ray of the gallbladder is of great value in this regard Other conditions such as diaphragmatic hernia, herpes zoster, cervical rib, thoracic tumor or aneurysm may also cause confusion However, a complete review of those features that characterize angina pectoris will generally lead to the correct diagnosis

Despite all our routine methods of study many patients with angina will show either nothing abnormal or there will be found only those minor alterations that are common in otherwise well people in the second half of

life Even when abnormalities are found they do not always characterize the anginal state The description of the type of distress is by far the most important feature from a diagnostic point of view Despite the great dependence on the subjective symptoms and notwithstanding the fact that they may be atypical and simulated by diseases other than angina, the diagnosis of angina pectoris by and large can be made with a high degree of accuracy

Prognosis

The prognosis in any case of angina pectoris is most uncertain Sudden and unexpected death which characterizes this disease may occur at any time It probably is just as well for all concerned that accurate predictions as to this fatal outcome cannot be made There is no other condition in which sudden and instant fatalities take place with the same degree of frequency In fact, when the diagnosis of angina pectoris is properly made the possibility and likelihood of this eventuality is always inferred A word about instant death may be appropriate at this point There are very few conditions that cause instant death Physicians are too prone to make the diagnosis of cerebral hemorrhage, stroke or apoplexy when a patient dies suddenly In my experience a rupture of a cerebral vessel from hypertension or cerebral aneurysm, or a cerebral embolism never kills instantly Such patients may suddenly become paralyzed or comatose but death, when it occurs, follows after several hours or more commonly in a few days Rarely a massive cerebral hemorrhage may kill in some minutes or in an hour I have seen death occur in eighty minutes in an apparently well woman 48 years old, who post mortem showed a ruptured cerebral arteriovenous aneurysm with massive intracerebral, subarachnoid and intraventricular hemorrhage Major peripheral emboli do not kill instantly Even a large pulmonary embolus presents a picture of sudden respiratory distress and suffocation and when fatal, death is apt to be delayed for a period of ten minutes or more Occasionally rupture of an aortic aneurysm may produce an instant fatality In fact most of the cases in which death occurs instantly, i e , within seconds or one or two minutes, are directly due to the heart and moreover are the result of a limited number of causes

Instant death may result from rupture of the ventricle This is by no means an uncommon event in those suffering from coronary heart disease and is also an occasional end result in subacute bacterial endocarditis Another cause is complete heart block Here the circulation may be quite adequate and compatible with a considerable degree of physical activity as long as the ventricles contract at a rate of 30 or more When an attack occurs in which there is failure of the ventricle to respond (Adams Stokes' seizure) unconsciousness results, and if the asystole lasts more than a few minutes consciousness does not return and the attack is fatal There is a very rare condition in which inhibition of the heart occurs It is not a matter of blocking of heart beats but rather that the beats do not arise, the pacemaker stops functioning This can be due to an oversensitive carotid sinus It is known that complete arrest of the heart may result for as long as ten seconds

by this mechanism and it is conceivable that rarely a fatality may occur The constant use of ephedrine in patients who present this peculiarity is of considerable value in preventing attacks Sudden fatalities occasionally occur during the administration of quinidine There have been a few observations that throw some light on this, in which it was found that occasionally quinidine causes inhibition or paralysis of the auricles Such cases have shown a temporary disappearance of the auricular complexes (Fig 49) If this same inhibition which has been shown to affect the auricles should affect the ventricles, the heart beat would suddenly stop In this way sudden death during quinidine administrations, not the result of other causes, may be explained A similar mechanism rarely follows excessive digitalis (Chap 21, Fig 48)

The last type of disorder that causes instant arrest of the circulation is ventricular fibrillation This must not be confused with fibrillation of the auricles In the latter condition the ventricles are contracting irregularly but actually expelling blood from the heart In the former the ventricles are essentially immobile producing no effective systole whatever and no flow of blood There are many reasons to believe that this mechanism is probably an important cause of sudden death In animal experimentation the heart is frequently seen to stop as a result of the onset of ventricular fibrillation after ligation of one of the coronary arteries This has generally been considered as the cause of the sudden type of death that characterizes angina pectoris Besides the above indirect experimental evidence in support of this explanation, there has been more direct proof of this point of view During some routine work it so happened that a patient suffering from angina pectoris was having an electrocardiogram taken He died in the attack and postmortem examination showed disease of the coronary arteries but no acute occlusion The electrocardiograms were typical of ventricular fibrillation This was direct proof that instant and unexpected death in angina pectoris could be brought on by ventricular fibrillation This mechanism is very likely a common cause of sudden death accompanying coronary artery disease When a patient with angina drops dead instantly one should not assume that he suffered from a fresh attack of coronary thrombosis Postmortem examination is likely to show coronary sclerosis and narrowing but no new fresh lesion When an acute thrombosis occurs he is more likely to survive for minutes hours or longer and live long enough to be seen by a physician

On rare occasions a surgeon is confronted with the problem of sudden arrest of the heart during an operation This may occur without the presence of organic heart disease In a few such instances electrocardiograms have shown that ventricular fibrillation was present Under these circumstances it is best to massage the heart manually and rhythmically and to spray the heart with 0 5 to 1 0 per cent procaine hydrochloride Five or 10 cc of the solution can also be injected intravenously Satisfactory recoveries have occurred with this technic

Because of the unpredictable occurrence of sudden death in cases of angina pectoris the prognosis in any given instance is very uncertain

Patients may die in the first attack or shortly after the onset of the first symptoms or may live in comparatively good health for over twenty years There is very little in the clinical features that enables the physician to distinguish one type from the other The average length of life after the first symptoms have developed is about four and one-half years for both sexes and the average age at death is 61 years for the males and 63 years for the females A more recent review by White has indicated more than seven years as the average length of life after the first symptom This increase, to a large extent, may be explained by more careful history taking If, on accurate questioning, an earlier date of onset of symptoms is elicited, the duration of angina will obviously be lengthened It was found that whether the tonsils had previously been removed or not made no difference in the course of the disease I believe that foci of infection have no important relation to this problem Slight differences in average life expectancy may be predicted on the basis of certain factors but on the whole such considerations do not help materially in estimating prognosis When angina develops before the age of 50 years the duration of the disease is about two years longer than when it first begins in later years Those who have a low blood pressure develop the disease a few years earlier and live a little longer after the onset of the disease than those with hypertension

There is one significant factor that aids in prognosis, i e , the hereditary one Those patients with angina pectoris whose parents died at an older age lived distinctly longer than those whose parents died at a younger age In this study of heredity it was also found that although males suffered from angina about four times as frequently as females, the inherited defect of vascular vulnerability was transmitted more prominently through the mothers of these patients than through their fathers In other words the females transmit this tendency more than do the males but are themselves less affected This I believe helps to explain the fact that in the latter decades of life, all vital statistics show that females outlive males by more than three years

The relation between angina pectoris and cardiac decompensation is of some interest Although it is generally stated that the former disappears when the latter develops, this is frequently not the case In fact there are instances in which both conditions are present simultaneously and the disappearance of the evidences of decompensation is accompanied by the disappearance of anginal attacks The presence of hypertension makes it more likely that congestive heart failure will develop sometime during the course of the angina The duration of life, however, after the onset of angina is one year longer in those who decompensate than in those who do not The great frequency of hypertension in women, therefore, explains the more common occurrence of cardiac decompensation in this sex when angina has been present

Obesity was not found to affect either the age of onset or the duration of angina pectoris I believe that if obesity per se acted deleteriously as a causative or aggravating factor the obese patients should have developed the disease earlier in life and died at a younger age than those with normal

or subnormal weights The fact that this was not found to be true suggests that obesity has no important direct relationship to angina pectoris but merely reflects the constitutional type that is more prone to the disease

Although electrocardiography is often of considerable value in the diagnosis of coronary artery disease its aid in prognosis is very slight In a study of over 100 cases in which electrocardiograms were taken it was found that the duration of the disease was one year longer in those with normal curves than the average, but inasmuch as angina developed earlier in life in the former than in the latter, these patients died at an earlier age than those with abnormal electrocardiograms The duration of life after the onset of angina was one year less in those with inverted T waves in Leads I or II than in those without such changes In a small number of instances showing other electrocardiographic changes, such as prolongation of the P-R interval or the QRS complex or QRS waves of low amplitude, the prognosis was slightly better than for the entire series It must be recalled that in many of the cases with these latter electrocardiographic abnormalities, the anginal features had become less prominent and the element of myocardial insufficiency with dyspnea and limitation in physical activities had become more important

It is of some interest to know what type of exitus is to be expected in patients with angina Approximately 50 per cent were found to die suddenly In addition about 30 per cent died of coronary thrombosis In these latter cases the patients were seen and lived long enough for the physician to make this diagnosis Many of the instant deaths in the former group no doubt were also due to coronary thrombosis That this is not invariably true is attested by the fact that in some cases of sudden death from angina pectoris there was no evidence of an acute thrombosis although atheromatous changes were noted in the coronary arteries Somewhat less than 10 per cent of the subjects died of congestive heart failure and the remainder died of miscellaneous causes such as cerebral hemorrhage, bronchopneumonia, cancer and other conditions The fact that there was such a high incidence of that type of death which one would expect in cases of angina pectoris is valid proof of the great accuracy of the clinical diagnosis

Treatment

The treatment of angina pectoris may be divided into two problems, the treatment of the individual attacks and the general care of the patient with the hope of diminishing the severity or frequency of recurring attacks Patients with angina should be instructed concerning the use of nitroglycerin This is the simplest and on the whole the most useful medication for the attack They should always carry some pills with them The dose should be $\frac{1}{100}$ grain or $\frac{1}{200}$ grain (0 6 or 0 3 mg) Although most patients find $\frac{1}{100}$ grain to be the suitable dose some will find either $\frac{1}{200}$ grain or even $\frac{1}{400}$ grain to be sufficient When nitroglycerin is first given it is wise to start with the smaller dose as occasionally the ill effects from the full dose so alarm the patients that they refuse to use it any more The pill should be placed under the tongue and should be dissolved in ten to twenty

seconds This should occur either spontaneously or as the result of sucking the tablet or crushing it between the teeth Tablets that are hard and dry and require minutes for complete dissolution should not be used The occurrence of a sensation of warmth or pounding in the head serves as a guide as to the potency of the pill The patient should be told to use it whenever he has the anginal distress unless it is so mild and of such short duration that it would be hard to know whether nitroglycerin really did much good Those suffering from angina generally learn before long whether the pill is helpful or not If its use appreciably diminishes either the duration or the severity of the attack it should be used The physician should further advise that nitroglycerin is to be used to prevent or anticipate attacks During the daily routine patients often learn that a particular act or set of circumstances frequently precipitates attacks Shaving in the morning, undressing at night, walking to the train, attending a business conference or the act of sexual intercourse are common precipitating factors and nitroglycerin can serve as a preventive in anticipation of such attacks Occasionally patients may find that taking a pill every two hours during the day may be helpful I have seen no ill effects resulting from the liberal use of nitroglycerin when it seemed indicated One woman consumed 1000 pills ($\frac{1}{100}$ grain) in one week without ill effects and there are many patients who have used 100 pills a week continuously for many years

It is very rare that amyl nitrite is needed or will accomplish what nitro glycerin will not The inhalation of amyl nitrite acts somewhat more quickly and more violently but is not so simple for daily routine use On the whole, amyl nitrite is used very little Alcohol in the form of suitable drinks is often of considerable help either during a spell or, better still, as a preventive Some individuals find ½ to 1 ounce of whisky taken before bedtime will prevent attacks from coming at night Finally the patient should either stop the activity that brings on the spell or he should at least slacken his pace Although it is well to give this advice it is generally unnecessary as sufferers from angina quickly learn that if they carry on and do not heed the distress it will continue or grow worse and if they stop still the pain lets up Some attacks are severe and protracted enough to require morphine

Quite recently an interesting relationship between carotid sinus stimula tion and anginal pain has been discovered It has been found that frequently the pain can be made to disappear in several seconds by massaging one or the other carotid sinus In an experience now comprising over one hundred episodes I have observed that whenever satisfactory slowing is produced the pain either promptly disappears or is markedly lessened Furthermore whenever no significant slowing of the heart results the pain is unaffected No such relief was obtained when the pain was due to a variety of other nonanginal causes, except in a rare case of functional pain The response was so distinctive that I believe this test has diagnostic value If well-marked slowing takes place from carotid stimulation while the patient has pain and it is not lessened, I believe that the pain is very unlikely anginal in nature In many instances the particular episode is completely relieved

and the pain does not return In others, as the heart rate speeds up again, the pain comes back Experience with pain of acute coronary thrombosis has been quite limited Some patients have shown no response, but in a few temporary alleviation of the pain while the heart slowed was observed

These observations, which can be made quite easily, tempt one to speculate concerning the mechanism of anginal pain At first it seemed that a neurogenic mechanism must be involved because the relief of pain was often almost instantaneous In one instance the pain disappeared in a few seconds actually while the heart was in asystole In this case a long asystole had resulted from carotid sinus stimulation One could readily explain this on the basis of a release of spasm in a coronary artery which obviously could take place instantly Another explanation however, may be that during the long single diastole, or in other cases when marked slowing occurs, the noxious catabolite that causes the pain (the unknown ' P element of Lewis) is washed out of the heart, while less or none of this irritating chemical is being produced This is plausible because during diastole the coronary circulation continues though there is no contraction of the heart, i e , oxygen supply continues but the production of breakdown products such as lactic acid is lessened In any event this maneuver can have some diagnostic and occasionally some therapeutic value

The second aspect of the treatment is much more difficult and less effective In most cases the transient attacks are readily controlled by the measures just mentioned, but how are we to diminish the recurrence of these attacks so that they will not prevent the patients from walking or working or conversing? There is no simple specific procedure that has proved very valuable for this purpose At the outset the question arises whether or not to enforce a period of strict rest in bed There have been two diametrically opposite views in relation to this method of treatment In one the importance of a period of bed rest for as long as several weeks or even months has been propounded In the other it has been maintained that this is not desirable but it has been recommended that the patient remain ambulatory and walk or exercise to the limit of his tolerance, i e , he should avoid the effort that brings on the distress if possible The very fact that such different views have been expressed and carried into practice by equally learned clinicians must mean that neither one can be universally applied nor that there can be any great difference in the results obtained Many considerations must be taken into account, especially economic ones One hesitates to put a man to bed for four weeks when the resulting loss of income would work great hardships on the family Particularly would this be true if such a procedure would jeopardize the patient s job or subsequent work Working men and women over 50 years of age are readily discharged by their employers only to have their places filled by younger employees and find it extremely difficult to replace themselves in industry These are matters that the physician must constantly keep in mind, for too often the social and economic status of patients is ruined without any significant gain in their health to compensate for this prevent

able loss On the other hand, when the person in question has no occupation, is retired from business or has sufficient means to enjoy the luxuries of life one would be more ready to try a period of prolonged rest

When attacks are recurring very frequently and are quite severe or when they actually prevent a person from attending to his work it is wise to try a period of bed rest for several weeks In some cases this alone improves the condition or at least renders the patient free from attacks or diminishes the frequency for a considerable period of time In some patients who suffer primarily from nocturnal attacks, placing 9 inch shock blocks under the head posts of the bed may eliminate attacks in bed The explanation of this beneficial effect of posture may be that return flow to the heart and therefore heart work may be decreased by this simple procedure Although the rigidity of the rest treatment will vary with different circumstances, in so far as possible it should provide complete mental and physical relaxation When kept at rest such patients should be permitted to leave their beds for movements of the bowels They should avoid all annoying experiences such as disturbing visitors and business conversations If there are pressing personal or business matters that are preying on the patient's mind which could be straightened out by a visit of a few hours at the office or by a conference, it is better to have it over with before instituting the rest treatment In other words, all reasonable measures should be employed to procure the rest that is desired It is generally better to do this in the patient's home or better still in a hospital than to send him off for a vacation, a procedure that is often ill-timed, expensive and ineffective

Another consideration in this treatment is the diet Many patients with angina pectoris are overweight and it will be found that when a period of bed rest is instituted, the semistarvation milk diet (Karell diet) will often prove very valuable This consists of 200 cc of milk four times a day and no other food, allowing a little more water for thirst This is a very low calorie diet and if continued for three days is apt to result in a feeling of weakness and hunger Accompanying this asthenic state attacks of angina which were occurring daily at rest, may disappear After a varying number of days of the Karell diet, small additional quantities of other food are gradually added Five small meals a day are preferable to three larger meals A high carbohydrate diet has been recommended by many and even the intravenous injection of concentrated glucose solution is practiced by some I have seen too little good come from the latter to advise its use although I frequently suggest the taking of sweets in the absence of diabetes When attacks of angina are related to hypoglycemia a high protein rather than a high carbohydrate diet is advisable, for this will insure a more even amount of sugar available in the blood stream Extra small feedings between meals also help to prevent the fall in blood sugar that is responsible for the attacks It is important that the diet contain the necessary vitamins and that it should be such that the patient does not gain weight It is well to advise a period of rest directly after meals whenever possible or at least to urge against any avoidable physical or mental effort on a full stomach, as attacks are very common under these circumstances

Whether the patient has been put to bed or remains ambulatory, a loss of weight is desirable in most cases When the amount of overweight is considerable I have seen marked improvement result from a gradual loss of 30 to 40 pounds in instances where nothing else was done, the patient remaining at work and taking no medication When the weight is normal at the start, a slight loss of weight might be advised

The relation of 'gas in stomach and anginal attacks has always been puzzling A great many patients are certain of the association so that they think they have stomach trouble or "indigestion and are never convinced that they have anything wrong with the heart They feel that gas brings on the attack and if they raise the gas the attack stops It is possible that gas is swallowed before or during the attack and later expelled On close examination it generally becomes clear that gas may be an accompaniment or result of the attack but that some physical or mental effort is the real precipitating cause and that gas with the patient at rest produces no distress Notwithstanding this, some would be a good deal more comfortable if the gastrointestinal tract were improved This is no simple matter and taxes our ingenuity to the utmost Proper bowel habits and whatever measures that will prevent constipation and straining at stool are indicated

One should not forget that peptic ulcer is by no means rare and disease of the gallbladder with or without stones is very common in patients suffering from angina When these additional diseases are present their symptoms must and can be distinguished in most instances from those resulting from the heart disease, and merit appropriate treatment It must not be expected that removing the gallstones will cure the patient of angina When this is supposed to have occurred I feel certain that the diagnosis of angina was incorrect I have had a great many patients with both conditions and found that when the gallbladder and stones had been removed surgically, those symptoms due to biliary disease disappeared but those due to the coronary artery disease persisted although they may have been ameliorated

When the question of smoking came up in former years I used to tell patients to smoke moderately, i e, not more than eight cigarettes or two cigars daily Now I am more inclined to urge omitting tobacco entirely It is generally believed that tobacco has vasoconstricting effects on the peripheral arteries It should always be avoided in a condition like Buerger s disease We now know that tobacco produces temporary depression of the T waves in the electrocardiograms in some individuals In fact, marked electrocardiographic changes characteristic of extensive myocardial infarction have been observed to appear shortly after smoking and entirely disappear within fifteen minutes This makes one think that a major coronary artery was temporarily in spasm producing local ischemia Although I have never seen tobacco per se produce angina in the sense that the disease would disappear on omitting its use, one does find patients who have fewer attacks if they do not smoke It therefore seems wise to curtail greatly or eliminate its use in cases of angina One should individualize in this matter One might wisely urge a man 40 years old who has been smoking 40 cigarettes a day t[illegible]

to smoke a pipe or a cigar after each meal In the first instance there is good possibility that the patient may live many years, while with the older individual the number of pleasurable experiences may have already become quite limited

A variety of drugs has been used with the purpose of diminishing the frequency and severity of attacks and of enabling the patient to do more work without discomfort The great diversification of these drugs is ample evidence of their inefficiency Potassium iodide (10 drops t i d) has long been used in all forms of vascular disease including angina pectoris The frequent reference to the value of this drug that is so prevalent in the older medical literature no doubt is due to the striking results that were obtained in some cases of masked hyperthyroidism and in syphilis when their etiologic significance was not understood Whether it is of any value in other cases is problematic and at least doubtful Digitalis has been used a good deal and it has been found that when angina is unassociated with dyspnea, general myocardial insufficiency or congestive failure it has been of no use There are some who believe it can aggravate angina pectoris It should be tried only when there is dyspnea apart from the anginal pain or when there is definite or suspicious evidence of congestive failure

The use of atropine was once advised by Sir Clifford Allbutt on the basis that the sudden fatal outcome that occurs in angina is due to vagal inhibition of the heart " There has been no proof either of this mechanism of death in angina or that atropine acts beneficially Inasmuch as some believe that an increase in heart rate is an important factor in the development of attacks and atropine can increase the heart rate by diminishing vagal tone the therapeutic indications for this drug are at least open to question

The common experience that all physicians have had in finding that emotional factors seem to precipitate attacks of angina has led to the use of sedatives Sir James Mackenzie believed that more good was to be obtained from bromides than from any other medication The purpose is to diminish the nervous receptivity or instability of the patient so that those reflexes that are involved in the production of the attack or in the sensation of the discomfort will be dampened For this purpose 1 gm (15 grains) of sodium or potassium bromide or 0 015 to 0 03 gm (¼ to ½ grain) of phenobarbital may be used three times a day I have witnessed occasional instances in which patients who were having frequent short attacks at rest have become entirely free from attacks promptly following such medication Unfortunately this is not the usual result At times it is distinctly valuable to render the patient seminarcotized for a day or two to break up a storm of recurrent attacks of angina using a preparation like sodium amytal, 0 2 gm (3 grains) every few hours In general I believe that the use of some form of sedatives, especially bromides, has a distinct place in the treatment of those patients who have frequent attacks of angina

Because of the importance of the psychic and nervous factor it is best for the physician to avoid the term 'angina pectoris *in talking to his* patients whenever possible It carries too great a dread in the mind of the

average lay person today When it is necessary to give explanations it is less shocking to state that the arteries within the heart are not so flexible or patent as normal or that the heart is simply tired

A sojourn to a warm climate is often advisable when circumstances permit this luxury It is a common experience that patients who can not walk a block during the winter in New England without pain are able to walk long distances in Florida Not only is it warmer but the weight of the clothes worn is less and the individuals are more relaxed

During recent years there has come into vogue a series of new preparations that are supposed to dilate the coronary arteries and increase the blood flow through the heart There is considerable experimental evidence in animals to show that these drugs do increase coronary flow Whether similar effects are produced in human beings and especially in those who already have atheromatous changes in the coronary arteries is another matter and more difficult to prove There is clinical evidence, however, although not all are agreed on this point, that some patients are more comfortable and can do more while taking the e drugs Of this group the common ones employed are diuretin (0 3 to 0 5 gm three times daily) aminophylline (0 1 to 0 2 gm three times daily), phyllicin (0 25 gm three times daily), quinidine sulfate (0 2 to 0 3 gm three times daily), theocalcin (0 3 gm three times daily), theominal (0 3 gm three times daily), theobrominal (0 3 gm three times daily) erythrol tetranitrate (0 03 to 0 06 gm three times daily), theobromine sodium acetate (thesodate) (0 5 gm three times daily), and others Physicians should employ those preparations that are least expensive and should omit them entirely if their observations indicate that no clinical improvement takes place It is well to try patients on such a preparation for one month, then omitting it for a month, alternating in such fashion and then deciding whether attacks are more or less trouble some on one regimen or another

Intramuscular injections of various tissue extracts have also been tried, but there is little valid evidence of their value Cobra venom injections have been employed, with some success, in stubborn cases when attacks were recurring frequently Although numerous other drugs have been used at one time or another they have generally proved disappointing These few which have just been discussed however, are worth a trial but great expectations should not be entertained from their administration Of these aminophylline, theobromine and their allied preparations and quinidine are the most promising

Very recently a new drug has been proposed by Anrep and his colleagues for the treatment of angina The preparation is called khellin which is derived from a plant (*Ammi visnaga*) that grows wild in Egypt Experimentally this has been found to increase coronary flow Only a few clinical reports as to its usefulness have appeared, but they seem to be favorable It is thought to act beneficially in angina and also in bronchial asthma, according to some observers I have had some experience with it and feel fairly well convinced that it is helpful In fact, it seems to do more good than any of the above mentioned oral preparations It is given in oral doses

(tablets) of 50 mg three times daily It can be given intramuscularly, but experience with this method is very limited In a well-controlled stu using placebos so that the observer did not know when the drug or wh the placebo was being given, about 50 per cent of the cases obtained defi improvement in the anginal pain The relief occurred within a day or t and persisted for as long as the drug was being taken and for a few d thereafter The difficulty was that in about one third of the cases the d caused ill effects such as nausea and dizziness, and had to be discontinu although in none were there any serious complications In some a smal dose, i e, 50 mg once or twice daily, could be tolerated and proved he ful Further observation will be necessary before final judgment can made, but at present it would appear that khellin (Eskell) has promis value, especially if the toxic effects can be eliminated

As happens in any chronic disease for which no specific effective tre ment is available, many bizarre therapeutic methods are suggested a often supported enthusiastically by their respective sponsors X-ray tre ment over the adrenals has been recommended on the basis that epinep rine acts as the trigger mechanism responsible for attacks of angina T tosterone propionate given intramuscularly has been heralded as a valua cure I have tried all these medications and have failed to be convinced t they have any real specific value The same may be said of vitamin E has been claimed by some that this produces striking benefit in card disease, especially angina pectoris Most critical observers have failed confirm this view Finally it is thought by some that a snug abdominal b applied so that pressure is exerted upwards, elevating the diaphragm, c prevent attacks of angina on effort This type of belt does help breath in cases of emphysema and possibly in cardiac patients with congesti failure, and also makes patients with enlarged liver more comfortable therefore may indirectly be of some value in selected cases of angina fact, more recent observations seem to confirm the original view of Ke that a definite minority of patients with angina are helped by an appropria abdominal belt

Surgical Procedures

The recognition of the limitations of ordinary medical treatment f many diseases, the lack of specific therapy and the rapid development surgical technic and newer surgical procedures have brought the gener surgeon into contact with problems that not so long ago were entirely co fined to the attention of the physician This has been true of practical all the organs of the body The most recent spheres of surgical intere have been the lungs, the brain and, last of all, the heart We are now seekin surgical aid in the treatment of cardiac disease because of the failure to gi our patients relief using the older methods The problem of heart disea in this respect is difficult for many reasons The morbid processes are f the most part progressive The fundamental cause that is *responsible f* these changes is generally poorly understood so that further progress the processes can be altered but very little Finally the obvious technic

difficulty of surgical manipulation of the interior of the heart has impeded the kind of therapeutic progress that has taken place in the treatment of disease of the kidneys, lungs and other organs Despite all this, attempts which at first might have appeared to be desperate have been made Early in this development simple traumatic wounds of the heart were repaired Some forty years ago Brauer introduced the operation of cardiolysis for adherent pericardium This formed the stepping stone for further surgical work that has proved of real value in some otherwise hopeless cases of pericardial disease Some years ago Cutler and Levine reported the first case of valvulotomy for mitral stenosis Although the first patient survived the operation and seemed to be somewhat improved, the great operative mortality of the procedure rendered it impracticable The surgeons likewise have been playing their role in attempts to relieve anginal pain

The first surgical attempts to relieve attacks of angina pectoris consisted in removing the cervical sympathetic ganglia This was first performed by Jonnesco following the suggestion made many years ago by the physiologist François Franck At first the entire chain consisting of the superior, middle and inferior ganglia including the stellate ganglia generally on the left side was removed Later a simpler operation in which only the superior ganglion was removed was proposed by Coffey and Brown The results obtained by these operations were rather variable It seemed that in about one half of the cases some relief was obtained but it was impossible to predict who would and who would not be improved I have had some patients that were greatly helped by this simple operation, including one who was enabled to walk miles without distress, and yet there were others who seemed equally promising subjects who were hardly relieved at all There was much controversial discussion concerning the rationale of these operations as the sensory and motor pathways to and fro from the human heart were not well understood The most recent point of view is that the logical method of interrupting the painful impulses is through the upper four or five dorsal ganglia and not through the cervical ganglia This has led to operations consisting of alcohol injections of the upper dorsal nerves or extirpation of these nerve roots The former type of operation carries with it almost no surgical mortality but often results in uncomfortable pains due to alcohol neuritis, whereas the latter is an elaborate surgical procedure which when carried out on patients with angina will entail an appreciable immediate operative risk At present alcohol injections are very rarely performed but interest in upper dorsal sympathectomy has been revived This latter operation can afford satisfactory relief in a large majority of cases that survive the operation Smithwick has performed a total dorsolumbar sympathectomy in a small number of cases of hypertension who also had angina, with no operative mortality and with very satisfactory clinical relief of the anginal pain It would seem that operation on the sympathetic nervous system still has its place in a limited well-selected group of patients suffering from intractable angina with or without hypertension

I recall an instance in which a boy 16 years of age was suffering from intolerable attacks of angina as a part of marked rheumatic aortic valvular

disease. The heart was markedly enlarged but there was no congestive heart failure. The boy's condition was pitiful for he had terrific attacks of anginal pain accompanied by palpitation. During these spells the blood pressure would rise markedly. The pain was very severe and would spread across the upper chest, making the boy cry and beg for relief. Nitroglycerin would alleviate and shorten the distress. These attacks occurred frequently even while the patient was at rest in bed. We contemplated surgical treatment as all medical methods had failed. The possible procedures that occurred to us were a simple decompression of the chest as there was marked cardiac hyperthrophy with a bulging of the precordium, a thyroidectomy or a complete left-sided cervical sympathectomy. It was decided to try the latter. The therapeutic result was most gratifying. The painful attacks disappeared. Although the boy had spells, they consisted of palpitation and the terrible agony that previously accompanied them was gone. Instead of being a most distressing, bedridden cardiac cripple he was able to return to school, gained weight and was happy. Similar favorable results in cases of severe angina accompanying aortic valvular disease have been reported following alcohol injections of the dorsal ganglia. I have had other instances in which, after such operations, patients who previously had been greatly handicapped were improved and enabled to walk and carry on without pain. The puzzling feature of all this is that the same operation often proved to be a complete failure in cases that seemed promising. It appears, however, that these operations may yet be indicated in selected cases for the relief of pain, although there is no evidence that they prolong life or that they prevent the subsequent occurrence of sudden death or coronary thrombosis.

Another development in the surgical relief of angina is complete thyroidectomy. Since 1932, when the operation was first performed, several hundred patients have been similarly operated upon in various parts of the world. Enough time has elapsed to form some estimate of the results obtained. The purpose of this procedure was to diminish the work of the heart and to eliminate any nervous or toxic effects that the thyroid gland has been suspected of producing. It was well known that subtotal thyroidectomy caused marked improvement in cases of thyrotoxicosis associated with angina. The operation was therefore applied to patients with a normal thyroid gland.

Observing the results obtained during the past years, I have come to the following conclusion. In severe angina the operation eliminated entirely or reduced the frequency of attacks in the great majority of cases. The surgical mortality was very small (2 to 5 per cent). There were serious complications following the operation in some instances. Postoperative tetany or aphonia was extremely rare. Occasionally distressing psychoses developed. The resulting mild but protracted myxedematous state led to definite disabilities such as rheumatic pains, feeling of coldness and fatigue, weakness, change in temperament and physical appearance, and other complaints. All these sequelae detracted from the value of the operation. Despite this, many patients were considerably helped and were thankful

for the relief of pain Others lived long enough to experience a return of anginal pain which was difficult or impossible to relieve Because of the frailties of human nature some of these latter forgot the fact that before the operation their pains were insufferable and that they begged for relief at any cost The final result is that at present I do not advise total thyroidectomy for angina pectoris

Finally different attempts have been made to increase the coronary circulation by surgical measures Beck sewed the left pectoral muscle to the pericardium overlying the left ventricle O Shaughnessy anastomosed a portion of the omentum through the diaphragm to the ventricle In both instances elaborate experimental work was first carried out in animals which seemed to show that new blood vessels developed in the heart muscle Later a considerable number of patients with coronary artery disease were subjected to these operations Also the left coronary sinus has been ligated and the nerve fibers resected from the coronary arteries by Fauteux in a small group of patients with coronary artery disease Here also the purpose was to encourage increased arterial supply to the myocardium

Another and simpler method of increasing blood supply to the myocardium has been employed by Thompson and Raisbeck They spray powdered talc over the visceral pericardium to induce adhesions of the two pericardial layers The report of their experiences seems encouraging

Following the idea used in the surgical treatment of cyanosed congenital cardiac patients, Beck has been trying to improve coronary artery flow by anastomosing the subclavian artery to the coronary sinus For the present these surgical procedures must be regarded as still in the experimental stage

It is a heartening sign that numerous attempts at surgical treatment for chronic heart disease are being made, for it is the last important organ for the surgeon to explore

CORONARY THROMBOSIS

The clinical recognition of acute coronary thrombosis forms one of the most interesting chapters in the history of medicine Although there were isolated instances in which a clinical antemortem diagnosis of occlusion of a coronary vessel was made, such as that by Hammer in 1878, and although there were other reports that touched upon the question in one way or another, the first publication that discussed the clinical features which might help us in differentiating an attack of coronary thrombosis from one of angina pectoris was by Obrastzow and Straschesko in 1910 A second one by Herrick appeared in 1912 It only then became clear that there were certain findings obtainable antemortem which differentiated the two conditions In fact Herrick first emphasized that the condition need not be fatal Before this there was a great deal known concerning the condition from a pathologic point of view but the clinical findings in the living patient remained entirely confused and obscured in terms like status anginosus and myomalacia cordis It is difficult to understand how the great clinicians of the past many of whom had had abundant postmortem experience, could have overlooked this problem, especially when it is appre-

disease The heart was markedly enlarged but there was no congestive heart failure The boy's condition was pitiful for he had terrific attacks of anginal pain accompanied by palpitation During these spells the blood pressure would rise markedly The pain was very severe and would spread across the upper chest, making the boy cry and beg for relief Nitroglycerin would alleviate and shorten the distress These attacks occurred frequently even while the patient was at rest in bed We contemplated surgical treatment as all medical methods had failed The possible procedures that occurred to us were a simple decompression of the chest as there was marked cardiac hyperthrophy with a bulging of the precordium, a thyroidectomy or a complete left sided cervical sympathectomy It was decided to try the latter The therapeutic result was most gratifying The painful attacks disappeared Although the boy had spells, they consisted of palpitation and the terrible agony that previously accompanied them was gone Instead of being a most distressing, bedridden cardiac cripple he was able to return to school, gained weight and was happy Similar favorable results in cases of severe angina accompanying aortic valvular disease have been reported following alcohol injections of the dorsal ganglia I have had other instances in which, after such operations, patients who previously had been greatly handicapped were improved and enabled to walk and carry on without pain The puzzling feature of all this is that the same operation often proved to be a complete failure in cases that seemed promising It appears, however, that these operations may yet be indicated in selected cases for the relief of pain, although there is no evidence that they prolong life or that they prevent the subsequent occurrence of sudden death or coronary thrombosis

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to prostatectomy and in old debilitated individuals who are in a state of shock for any reason Attacks of cerebral failure may occur as a result of the same mechanism that has just been discussed

Coronary thrombosis is related to angina pectoris in much the same way as an occlusion of a vessel of the leg with gangrene is related to intermittent claudication The anginal state may be regarded as a transitory one, leaving the heart in practically the same condition after an attack as before When a partial or complete occlusion occurs, the muscle supplied by that vessel suffers to a lesser or greater degree Sometime during the life of those suffering from angina a thrombosis of a coronary artery is apt to occur This is the common cause of death in angina, although not a necessary one There are a few cases of angina even among those in which death occurs instantly, in which no acute thrombus can be seen Such hearts in my experience have almost always shown significant atheromatous changes in the coronary arteries In fact, I have seen but one case of angina in which the coronary arteries were normal except when there was one of those associated conditions discussed above, like aortic valvular disease, that adequately explained the condition Also, there have been a few cases of angina reported by others in which the coronary arteries were regarded as normal

It has always been puzzling to explain the precipitating causes of an acute thrombosis There is both clinical and pathologic evidence to make one believe that it occurs at the site of a previous sclerotic or atheromatous area in a coronary vessel The occlusion, partial or complete, then occurs rather suddenly It is not due to an embolus dislodged elsewhere, for coronary emboli are very rare and apt to be associated with vegetation at or near the aortic valve Do further platelet thrombi develop on the roughened surface of the vessel which lead to greater narrowing of the channel? This seems reasonable but does not quite explain the extreme suddenness of some of the attacks Or does the final occlusion result from the rupture of a miliary subendothelial atheromatous "abscess as some experimental studies of Leary suggest? The latter hypothesis would explain the abruptness of the symptoms better than the former Another possible mechanism that has been postulated is that minute hemorrhages first occur within the layers of the wall of the coronary arteries and these are followed by changes in the vessel wall such as narrowing and thrombosis Possibly all mechanisms are involved in different cases

A new method of studying the coronary arteries by a special type of injection devised by Schlesinger has thrown considerable light on this problem It has revealed that many thromboses occur that are not recognized in life and that are not accompanied by the clinical features of an acute attack to be described shortly It has also been found that thromboses occur without infarction and that infarction can also take place without thrombosis Most hearts have shown two or more occlusions, even as many as ten in rare instances This technic has revealed many new, interesting and important clinical pathologic correlations

Another perplexing aspect of coronary thrombosis is the frequency with which a particular part of the coronary system is involved A common site

is in the descending branch of the left coronary artery about 1 inch from its origin Sclerotic changes and thrombosis are so common at this particular spot as to make one suspect that a mechanical factor is involved Is it possible that as the heart contracts there is a greater bend or torsion of the artery at this point? If this were so it could throw some light on the hereditary factor that is so striking in this disease We do inherit specific anatomic characteristics such as the color of the iris and configuration of the nose May not those several members of a family with early coronary artery disease inherit a peculiar architecture of these vessels which puts them under greater mechanical strain as a result of the normal twisting and bending that goes on with the motion of the heart? This presents an anatomic and physical problem about which we have entirely too few data

The Clinical Picture

Before taking up the clinical aspects of acute coronary thrombosis it will be helpful to review briefly the pathologic process that is taking place When a coronary vessel becomes partially or completely occluded the area of heart muscle supplied by that vessel becomes infarcted Early in the process there is extravasation of blood, then muscle fiber necrosis and finally repair by scar tissue If the involved area extends to the periphery a localized aseptic pericarditis may develop, and if it extends inwardly the endocardium may be involved, resulting in a large secondary ventricular mural thrombus attached to the area of infarction The local area of necrosis may become sufficiently softened to perforate during the early days or the wall may be weakened to such an extent that a localized aneurysm of the ventricle results When satisfactory healing takes place due to the anastomosis of collateral vessels, which is much more abundant in the heart than was formerly thought, a healthy scar forms This simple review enables one to picture some of the clinical events that occur

Attacks of coronary thrombosis seem to occur more frequently at rest than during effort Furthermore, the patient may become restless and walk around or pace the floor instead of remaining still In this respect it differs from angina pectoris It is a common experience to find that the patient is aroused from sleep with the attack On close questioning many will confess that during the preceding day or two they had not felt quite so well and may have had more or less milder discomfort in the chest Occasionally one may suspect that a coronary thrombosis is impending when a patient who previously had ordinary attacks of angina promptly relieved by rest or nitroglycerin suddenly starts having attacks without effort, lasting a half hour or so and not responding to nitroglycerin, also, if similar severe symptoms occur without preceding angina, an outspoken coronary attack not infrequently follows in a few days

In its typical form the pain of coronary thrombosis becomes very severe almost unbearable The location and severity of the pain varies a great deal in different cases just as there is a striking variability of most of the clinical features of the disease Although no single sign or symptom is found in all cases, there generally is enough evidence of one form or another to make a

fairly definite diagnosis The amount of pain may vary from none at all to the most severe agony any mortal can suffer It takes the form of a pressure or terrible crushing or squeezing sensation Its location most often is in the middle of the chest centering around the sternum, or between the two nipples It can either begin in or even be limited to the upper epigastrium near the ensiform *It often radiates to the upper midback, shoulders and* arms It must be borne in mind that in some the distress is not very marked and in fact there are a few who are not prevented from carrying on their activities throughout an attack The duration of the pain is also variable, lasting from an hour or two to several days In some it comes in waves, each lasting about fifteen minutes until it becomes continuous Generally after six or twelve hours the severity has abated although a milder soreness persists *Not uncommonly after the pain has subsided it returns in varying* degrees for several days or longer

Breathlessness may be the presenting complaint particularly in those occasional instances in which pain is very slight or absent Although dyspnea is not present in most cases there are a few in which the clinical picture is one of acute pulmonary edema Even when the respiratory symptoms are not so fulminating, orthopnea or other evidence of embarrassed breathing may be apparent

In the great majority of instances there is a peculiar and rather characteristic appearance of the patient during the height of the attack He seems to be in shock The skin is cold, moist and takes on a gray ashen color The face is anxious and one is quickly aware that something profound has happened In the early hours and days sweating is often very marked, resulting in dehydration and a loss of salt from the body In fact a very moist skin may persist for weeks and gives one the impression that the underlying process of myocardial infarction is still active or insecure In some atypical cases a moist skin may be the first clue that the illness which was painless and consisted mainly of weakness is due to an acute coronary thrombosis Great weakness is often very striking even when there are but few other complaints With this there is generally a fall in the blood pressure This fall occurs in varying relation to the onset of the attack Sometimes the first reading that the physician obtains is extremely low or the patient may already be pulseless Not infrequently the blood pressure is still elevated during the first few hours and falls subsequently On very rare occasions no significant change takes place at all In the typical case the systolic pressure that was around 170 mm gradually falls to 110 mm, although this change may take place over several days With the progress of the case it may return to its previous level or be permanently lower to a greater or lesser extent Practically always the pressure returns approximately to the original level *during the following year or two*

Gastrointestinal symptoms are often prominent and may be troublesome from a diagnostic or from a therapeutic point of view The abdominal pain and tenderness which occur in a few cases resemble an acute surgical condition Even slight icterus may be present Nausea, vomiting and distention may be distressing and at times resemble the symptoms of acute intestinal

obstruction Vomiting is very common during the first hours of the attack and may become even more prominent after the frequent injections of morphine are given which are necessary to control the pain Occasionally annoying hiccoughs develop Usually all these symptoms disappear after one to several days

A slight fever and leukocytosis are very common accompaniments of coronary thrombosis They both require some time to develop and are generally present in twenty-four to forty eight hours, although they may be found at times as soon as six hours after the onset The fever is often overlooked if the temperature is taken by mouth as these patients are in shock and the periphery of the body may be actually cold in the presence of a true fever This error will be avoided if a rectal temperature is taken I have often found the latter to be ± 101 F when the mouth temperature was only 98° F The slight fever lasts from three to seven days, rarely longer The leukocytosis ranges around 15,000 to 20,000 and also lasts several days The sedimentation rate of the erythrocytes is also increased, probably as a result of the myocardial infarction This increase is apt to occur a few days after the onset of the attack and may persist for two weeks or more even after the fever and leukocytosis have disappeared Occasionally the sedimentation rate may indirectly aid in differentiating an attack of coronary thrombosis from one of angina pectoris or other conditions or may help in the decision as to whether an extension or aggravation of the myocardial infarction has taken place

The findings on examination of the heart are variable The sounds generally diminish strikingly in intensity so that at times they are almost inaudible The rate becomes accelerated to about 100 to 120 and in many instances a definite gallop rhythm can be heard Just as is true of all these individual features, there are numerous exceptions Occasionally there is no acceleration of the heart rate A small number of patients will show a transient pericardial friction rub When this occurs it is extremely helpful in diagnosis but its development will depend upon the location and extent of the myocardial infarction Almost any of the disturbances in rhythm with which we are familiar may develop during the early days following an acute coronary thrombosis When they do occur most of them are transient and the normal rhythm returns Of the important arrhythmias, transient auricular fibrillation is most common Auricular flutter is rare but ventricular tachycardia is not uncommon Extrasystoles are also common Some patients will show partial and a few will develop complete heart block even with Adams-Stokes attacks of syncope Conduction defects are more apt to develop with posterior lesions The apex impulse disappears as a result of the feeble contraction Although one might think that with such a profound injury to the cardiac mechanism dilatation would occur, x-ray studies have shown no such change

The examination of the lungs will frequently show some rales at the bases, especially on the right side Occasionally typical evidence of acute pulmonary edema will be found with moist or bubbling rales all over the chest In fact, an attack of coronary thrombosis may be ushered in by this

type of onset even without chest pain Sudden dyspnea may, therefore, replace sudden pain as the presenting complaint Cheyne-Stokes breathing is very common even when there is no subjective respiratory distress The abdominal examination is generally not remarkable, although in a few cases the findings resemble an acute surgical condition for there may be tenderness and muscular spasm in the epigastrium or right upper quadrant In some cases at least these findings are due to an acute passive congestion and enlargement of the liver with the accompanying tension on the liver capsule In one case in which the abdomen was explored as a result of a mistaken diagnosis this was observed

The urinary findings deserve some mention Many patients will show marked oliguria and some will void practically no urine for twelve to twenty-four hours This is the result of the low blood pressure and the state of shock The urine that is voided is concentrated and may show an appreciable amount of albumin and numerous cells and casts Glycosuria is very common, first because so many diabetics develop coronary thrombosis and because others who never had glycosuria before may temporarily show sugar in the urine as part of the coronary attack Some of the latter are possibly mild or potential diabetics but it seems that in others, who temporarily show glycosuria, it is entirely precipitated by the heart attack What is important to bear in mind is that the urinary findings indicative of renal disease need give us no great concern as they always clear up if the circulation improves The same may be said of the glycosuria It rarely if ever requires insulin during the acute stages In general it is better to disregard the diabetes unless, as very rarely occurs there is a significant acidosis as shown by a lowering of the carbon dioxide combining power of the blood

Electrocardiography has been a most important aid in the diagnosis of coronary thrombosis Although Herrick very early in his work suspected that certain electrocardiographic changes might possibly be the result of coronary thrombosis, it was Pardee who first established the fact that during the early hours or days after an attack, the ventricular complex may take on a peculiar form This consisted of a high take-off of the T wave from the QRS complex before the latter reaches the isoelectric line in one or another of the customary three leads This does away with the normal short flat interval between the initial and final phases of the ventricular complex It has also been found that rapid alterations in the complexes occur during the succeeding days or weeks following an attack The T waves become rounded and dipped (cove shaped) and finally peculiarly inverted (Chap 21) At any one particular time these tracings may appear essentially normal but if a series of several electrocardiograms is taken during the first two weeks, most if not all will show definite or at least suspicious alterations The introduction of precordial leads by Wilson Wolferth and Wood has further increased the usefulness of electrocardiography, for there are some instances in which the area of infarction is so located that the customary limb leads fail to show any significant aberrations while the chest lead will bring forth definite evidence of heart muscle injury

Electrocardiography has not only been valuable in diagnosis, but has enabled us to predict in many instances the exact location of the area of infarction. Injuries to the anterior part of the heart generally occurring in the lower lateral portion of the left ventricle near the apex and resulting from a thrombosis of the descending branch of the left coronary artery are associated with one type of electrocardiogram. Similar injuries to the posterior part of the ventricle, either resulting from a thrombosis of the circumflex branch of the left coronary artery or of the right coronary artery, produce a different set of changes. In the former the high take-off of the T wave with subsequent inversion of the T wave and the appearance of a Q wave occur in Lead I. In the latter type these similar changes occur in Lead III. Wilson has indeed shown that while the changes in the T wave are often transient with even complete restoration of the normal appearing upright T wave, the changes in the initial ventricular complexes, notably the Q wave, are apt to be permanent. In this way one may be enabled to suspect months or years after an attack that one previously had taken place. Further electrocardiographic details will be taken up in Chapter 21. All forms of conduction disturbance are found, such as partial or complete heart block and bundle branch block. Also in some the ventricular complexes are of unusually low amplitude. Unless the changes are quite characteristic, caution must be exercised in accepting alterations in the complexes as unequivocal evidence of myocardial infarction, as they may occur in a variety of other conditions such as pulmonary embolism, acute dissecting aneurysm, acute nephritis, uremia and rheumatic carditis.

In an uncomplicated case of coronary thrombosis of moderate severity the patient is apt to become free from pain after the first day and thereafter to remain quite comfortable. In other patients pain returns irregularly over a much longer time. Those whose respiratory function becomes embarrassed may have marked dyspnea and orthopnea. The various irregularities of the heart previously mentioned may suddenly change the clinical status and require special attention. At any time during this illness sudden death can occur either as a result of ventricular fibrillation or rupture of the softened infarcted area of the ventricle or possibly from complete heart block. Ruptures do not usually occur until about the fourth to seventh day and take place in those areas that have little if any fibrosis. It is more likely to occur in those cases in which there has not been much of a fall in blood pressure.

When the infarction involves the ventricular septum and is extensive it may rupture. The main evidence of this is a sudden appearance of a loud systolic murmur over the midprecordium and near the apex. With this complication there is likely to occur a sudden change for the worse with collapse. Bernheim's syndrome may develop, i.e., right-sided heart failure with increased venous pressure resulting from bulging of the interventricular septum into the right ventricular cavity.

Another type of complication is embolism. In many of these cases a fresh mural thrombus develops in one of the ventricles, generally the left, con-

tiguous to the area of infarction Portions of this thrombus may become dislodged and occlude other vessels producing secondary embolic lesions If they come from the left ventricle hemiplegia, renal, splenic or mesenteric infarcts may result or gangrene of one of the extremities If they arise in the right ventricle, pulmonary embolism or infarction occurs These are most apt to take place after the first few days The pericarditis that occasionally is found requires no particular concern as it almost never results in empyema or pericardial effusion It is evident, therefore, that the exact course which any individual case may take can vary greatly

Fatalities may take place with great suddenness even when everything seems to be going most favorably In other patients there is a gradual weakening of the pulse with a low blood pressure, marked weakness and a quiet, peaceful end In some there is a great deal of dyspnea and air hunger

When recovery takes place we also have different sequelae to be anticipated There are some who having suffered from angina pectoris, either have no symptoms at all after an attack of coronary thrombosis or have much less trouble than they had prior to the attack These patients are apt to be the ones who previously had hypertension in whom the pressure becomes permanently lowered after an occlusion of one of the coronary arteries In others anginal attacks return with the same frequency and in some angina pectoris may appear for the first time after a coronary attack There is a large group which never having shown any dyspnea or congestive heart failure, first begins to manifest these symptoms after an attack of coronary thrombosis Occasionally this occurs during the first week following the attack but more commonly the patient becomes ambulatory and only months or years later begins to have general circulatory failure

A localized aneurysm of the left ventricle with thinning and weakening of the wall occasionally results from a previous infarction Curiously enough rupture of the ventricle which is by no means rare during the early days of an acute attack, rarely if ever occurs at the site of such chronic fibrosed ventricular aneurysms They are present after recovery has taken place and the patient is ambulatory, and the condition is compatible with a fairly satisfactory state of the circulation The diagnosis of ventricular *aneurysm will rest on a previous history of myocardial infarction*, the finding of a visible and palpable apex impulse well inside the outer border of dullness, a diminished first heart sound certain suggestive electrocardiographic changes (Fig 159) and x ray examination The latter will show a localized bulge and on fluoroscopic or kymographic examination this bulge will be observed to expand outward with systole as the neighboring musculature contracts inward

Prognosis

The prognosis for patients with acute coronary thrombosis with myocardial infarction is variable When large series of these patients were first studied it seemed that about 50 per cent recovered and the other 50 per cent died during the acute attack The true figures are now much more

favorable because in the former studies only classical, easily recognized cases were included Now, with more advanced methods of diagnosis, many milder cases can be included and the immediate mortality will vary between 15 and 25 per cent, depending on the accuracy of the diagnosis

In an extensive review of the immediate mortality certain points of interest came to light Anterior and posterior lesions were equally serious The mortality was lower if significant electrocardiographic changes were slight or absent, was slightly higher for women than men and was definitely higher in older than younger individuals A previous history of angina improved the immediate prognosis, while a preexisting hypertension made it worse A marked fall in blood pressure, especially if the level was maintained below 80 mm for some time, made the outlook quite grave The severity of dyspnea was more ominous than the degree of pain In general, *the immediate mortality was greater if the customary clinical features of* acute coronary thrombosis, i e, a higher fever, more rapid pulse, greater *greater leukocytosis and more profound shock, were more prominent*

After recovery from an acute coronary thrombosis had taken place the duration of life was found to vary greatly, the average being about three and one-half years There was little difference in the length of survival between subjects with anterior and those with posterior infarctions Those with only minor alterations in the electrocardiograms lived longer One fourth died in one year, one half in two years, three fourths in five years and the remainder at varying intervals up to more than twenty five years Two thirds had angina some time after the attack of coronary thrombosis and about one fourth developed congestive failure The latter complication occurred more frequently with anterior than with posterior infarction About 30 per cent resumed essentially full duties for varying lengths of time and the remainder were more or less restricted The survival period for women was much shorter than for men and it was much longer for younger subjects than for older ones

As an illustration of the difficulties in prognosis the following experience is of interest A physician had a severe attack of coronary thrombosis at the age of 45 During the subsequent fourteen years he had several more attacks of coronary thrombosis and many attacks of paroxysmal ventricular tachycardia On more than one occasion he was in profound shock semiconscious and pulseless, with a heart rate of 200 or more With the first attack and during most of these years a definite gallop rhythm was present He has not shown evidence of significant congestive failure and there has been no dyspnea He is still ambulatory but has frequent attacks of angina which are promptly relieved by nitroglycerin Incidentally, he persists in smoking ten cigars daily, eats two eggs every morning and is quite happy

The knowledge that some can recover and carry on in good health for a long time, even fifteen years or more, permits us to encourage our patients Furthermore, satisfactory recovery from a second and even multiple attacks occurs but with increasing rarity In general the prognosis should be guarded but always hopeful for even in the face of an extremely severe and desperate attack satisfactory recovery may take place

Differential Diagnosis

There are several conditions that may at times become easily confused with coronary thrombosis Of first importance are the conditions that resemble acute surgical states of the abdomen such as gallstone colic, perforated peptic ulcer, acute pancreatitis, acute appendicitis and acute intestinal obstruction All the diagnostic methods available may be required to arrive at the correct diagnosis in such cases, and despite care there will be rare instances in which errors will be made The presence of dyspnea with the attack and the radiation of the pain to the sternum or to the arms may be helpful in differentiation The electrocardiograms may be the turning point on which a diagnosis will rest There is also the opposite danger of overlooking an acute surgical condition requiring immediate abdominal operation in our enthusiasm to detect instances of coronary thrombosis, for it must be borne in mind that patients with known organic heart disease, as well as those who are only suspected of having it, may also have these very surgical conditions that require an operation Under certain circumstances, when the diagnosis is in doubt, it probably would be safer to explore the abdomen even at the risk of an unnecessary operation rather than to overlook a perforation of the stomach

Another condition that may closely simulate acute coronary thrombosis is pulmonary embolism or pulmonary infarction (acute cor pulmonale) In both there may be sudden circulatory collapse dyspnea, a rapid thready pulse and cyanosis Pain in the chest is less common with pulmonary embolism, although it does occur and is particularly disconcerting when it takes place on the left side On the other hand, pain may be absent in both conditions During the early hours after a pulmonary embolism there is no hemoptysis and examination of the lungs is apt to be of no help in diagnosis Both conditions may occur after surgical operations but it will help to simplify matters if a cause for pulmonary embolism such as phlebitis can be detected Distention of the veins of the neck, although not an absolutely distinguishing physical finding is more likely to be present with a pulmonary than with a coronary attack In pulmonary embolism the pulmonary second sound is often accentuated and there is a systolic murmur in this area probably due to the dilated pulmonary artery and the increased pulmonary pressure proximal to the embolus Finally, the electrocardiograms may prove valuable in diagnosis Minor changes in the ventricular complexes are common to both, but outspoken alterations like a very high take-off of the T wave or a sharp rounded inversion of the T wave occur only with cardiac infarction Occasionally the electrocardiograms are sufficiently distinctive to make a fairly definite diagnosis of acute pulmonary embolism (Chap 21, Figs 161 to 166)

Two experiences, almost identical in detail, that had important medico legal implications are of interest in this regard A man about 45 years old was accidentally hit in the ankle while at work He limped around for the rest of the day having mild pain in the leg A few days later an x ray revealed a slight fracture The leg was put in a cast and the man put at rest Two weeks later he suffered an attack which was diagnosed acute coronary

thrombosis with posterior myocardial infarction He died a few days later and no autopsy was performed Three months later I received the above information and was asked by the widow's attorney to help in the litigation that was going on Not wishing to appear in court I was ready to express an opinion in writing I stated that if the man died of a coronary thrombosis it was questionable whether there was any relation between the original accident and the death three weeks later However, I suspected that the correct diagnosis was a sudden pulmonary embolism from a thrombophlebitis of the right leg and if that were true death could be regarded as a direct result of the accident In that case the trauma, the cast and the immobility caused the thrombophlebitis and the dislodgement of a clot caused the fatal pulmonary embolism I therefore urged that the body be disinterred and an autopsy be performed This was done and the predicted findings were confirmed The heart was normal, there was a large pulmonary embolism and a thrombophlebitis of the leg was found The second experience was exactly similar to the above In both instances the widows rightly obtained the compensation that was their due, which otherwise would have been denied them

The lessons to be drawn from these two experiences are (1) that the physician should think first of pulmonary embolism rather than coronary thrombosis if some thoracic catastrophy follows a trauma, and (2) that electrocardiograms of acute cor pulmonale may resemble those of posterior myocardial infarction Furthermore, it is apparently valuable at times to perform an autopsy even months after burial

In reviewing the differential diagnosis I need but recall errors that have occurred, some of which I have made myself In one instance some years ago I made the diagnosis of coronary thrombosis because of sudden suffocation and collapse occurring in a man 60 years of age The patient seemed to be in shock with a rapid thready pulse Although there was very little pain I thought he had had a coronary accident It was not until the next day that I realized that one side of the chest was not expanding with respiration and there were practically no breath sounds on that side He had a complete pneumothorax from which he recovered very satisfactorily

Another more common difficulty in diagnosis is the differentiation of pneumonia and coronary thrombosis *The fairly acute onset of chest* pain, cough, dyspnea, cyanosis, the development of fever, leukocytosis, rapid pulse and rales in the lungs occur in both conditions There are no significant alterations in the electrocardiograms in pneumonia, however This differential diagnosis at times is very difficult and I recall erring in both directions, considering a case one of pneumonia when it was coronary thrombosis and in another making the opposite mistake Electrocardiograms may serve to differentiate the two No doubt many elderly patients considered as having pneumonia really have suffered an attack of coronary thrombosis

Diabetic acidosis and coma may be simulated by acute coronary thrombosis Inasmuch as the latter often occurs in diabetic patients glycosuria is very common There may also be an associated acido is with diacetic acid

in the urine and a lowered carbon dioxide combining power of the blood When such findings accompany a state of semistupor or complete unconsciousness one is strongly tempted to regard the condition as diabetic coma All this, however, can result from an attack of coronary thrombosis Inasmuch as insulin may be harmful to patients with coronary artery disease great care must be exercised in differentiating the two conditions If the blood sugar is not high, insulin should not be given and even when found to be elevated only small doses should be used unless it is certain that diabetic coma is present In general I have very rarely found it necessary to use insulin in such patients

Hamman has called attention to a condition that may closely simulate acute coronary thrombosis He called it ' spontaneous interstitial emphysema of the lungs Many of us must have confused these two conditions in the past Such patients are suddenly stricken with violent pain over the precordium with radiation to the left shoulder and left arm and there may be an acceleration of the pulse, a leukocytosis and slight fever All this results from the rupture of an air sac in the lung that dissects its way along the bronchi and blood vessels and infiltrates the mediastinal tissues The air may extend in the subcutaneous tissue to the neck or may reach the pleural space, producing a pneumothorax The most important and characteristic finding is the presence of unusual clicking grunting or crunching sounds synchronous with the heart beat These sounds may be influenced by respiration and may be entirely absent with the patient flat on his back only to be brought out in the left lateral position In some, the x-ray will show air in the left pleural cavity or in the anterior mediastinal spaces This condition is essentially benign, as complete recovery is apt to occur The only treatment necessary is sedation for the pain and a brief period of rest Its importance lies in the fact that it needs to be differentiated from coronary thrombosis and it impels us to watch carefully for these unusual auscultatory findings It also must not be confused with noises that are rarely heard in cases of diaphragmatic hernia Such splashing heart noises result from the beating of the heart against the stomach or intestines, which may lie in the chest

There is a group of patients in whom the early symptoms of the acute stage are so mild that they are disregarded and the subjects present themselves as medical problems only after an embolus has occurred, particularly when a hemiplegia results Some cases that are diagnosed cerebral hemorrhage fall into this group The possibility that a hemiplegia may be due to an embolus dislodged from a left ventricular mural thrombus following a coronary thrombosis should always be considered when it occurs in a patient who has a low blood pressure Even in elderly patients cerebral hemorrhages of the ordinary type, excluding those resulting from aneurysms of the cerebral vessels, rarely if ever occur without hypertension A consideration of all the diagnostic points may be necessary in order to arrive at the correct diagnosis

There are atypical cases of coronary thrombosis in which extensive experience will be necessary to avoid making erroneous diagnoses In particular

is this true of the occasional instances in which pain does not occur Here there is apt to be sudden breathlessness and a feeling of exhaustion In such cases the dyspnea will be found to be out of proportion to other evidences of heart failure When this is the case, a low blood pressure, if known to be high before, together with other features such as changes in the electrocardiograms will give one the proper clue

When the coronary vessels are slowly narrowing, infarctions of the heart may occur without any acute episode This may take place in patients who have progressive heart failure and are regarded as suffering from chronic myocarditis Even here a proper survey of all the data, especially obtaining a history of early angina pectoris and the study of the electrocardiograms, may enable us to anticipate the true pathologic state

Considerable interest has developed in the diagnosis of dissecting aortic aneurysm which is often confused with acute coronary thrombosis The pain in the fomer comes with even greater suddenness than in the latter The pain in the chest is crushing may extend through to the back and at times even to the legs Hypertension is almost invariably found and it tends to persist after the attack Fever and leukocytosis develop but there are none of the irregularities of the heart or a friction rub that occur in coronary thrombosis Although the electrocardiograms in most cases remain unchanged and fail to show significant alterations in the ventricular complexes, T wave changes resembling myocardial infarction may appear as a result of pressure on one of the coronary arteries by the aneurysm In fact, some of the peculiar findings in this condition will depend on the location of the dissecting aneurysm and the direction which the dissection takes After splitting the wall of the aorta, the aneurysm and blood clot may extend down the abdominal aorta even to the iliac vessels In its course it may compress any of the arteries that are given off from the aorta and produce a variety of symptoms Giddiness and blurring of vision may result from the effects on the carotid arteries, anuria or hematuria from involvement of the renal vessels and pain and numbness in the legs from occlusion of the common iliac artery Paralysis of the limbs may develop from involvement of the spinal cord The peripheral pulse in one or another of the limbs of the body may disappear Sudden death frequently occurs from rupture of the aneurysm Although the two conditions have many clinical features in common, careful consideration of the finer differential points involved, especially the direct findings in the heart, will generally suffice to distinguish coronary thrombosis from dissecting aneurysm

Dissection and rupture of the aorta, often arteriosclerotic in type, may take place in its abdominal portion The symptoms can then be quite baffling and resemble acute pancreatitis, perforated peptic ulcer or an acute gallbladder attack There may be fever, a brisk leukocytosis and pain in the right upper quadrant and back The dissection may rupture into the pleural cavity

Incomplete tear or rupture of the aorta also occurs that may or may not be followed by dissection It is extremely difficult to recognize this condition clinically It may account for attacks of choking or suffocation or mid

chest pain occurring in patients with hypertension These tears are generally horizontal and when they are present quite close to a commissure of the aortic valve, it appears that the valve may sag and result in aortic insufficiency This is thought to be the explanation of an aortic systolic and diastolic murmur that may develop in some patients with incomplete rupture of the aorta Healing of these tears can take place or dissection may develop hours, days or even months later

With the great increase in knowledge concerning coronary thrombosis that has taken place in recent years, the diagnosis is now being made too frequently Whenever the evidence is not entirely convincing one should search for other conditions Apart from gallbladder disease and gastro intestinal disturbances already mentioned, bleeding peptic ulcer and diaphragmatic hernia must be considered The shock, weakness, rapid pulse, and fall in blood pressure resulting from hemorrhage can resemble a painless coronary thrombosis Similarly the peculiar distress in the chest that may occur with a diaphragmatic hernia, especially in women, may simulate a coronary attack In neither case, however, will there be found reliable evidence of myocardial infarction Herpes zoster, arthritis of the spine, carcinoma of the lung and syphilitic aneurysm of the aorta are other conditions not to be confused with coronary thrombosis The list of diseases that may enter into a differential diagnosis is almost endless, as is well illustrated by an experience in which coronary thrombosis was confused with the very rare condition called diaphragmatic flutter

Finally the differentiation of simple angina pectoris and coronary thrombosis needs consideration Generally this is not difficult All the features discussed in the preceding pages under these two headings help to distin guish the one from the other Although most attacks of angina last only a few minutes, some continue for fifteen minutes or more It is not always possible to detect evidence of coronary thrombosis when attacks last longer, although some of these no doubt result in myocardial infarction In many cases, on postmortem examination, there have been noted several isolated areas of fibrosis from old infarction, in which a corresponding number of attacks cannot be distinguished from a clinical point of view Furthermore I see patients with typical angina pectoris in whom I can obtain no evidence whatever of an attack resembling typical coronary thrombosis who yet show definite evidence in the electrocardiograms of a previous myocardial infarc tion The term acute coronary insufficiency may well be applied to attacks of prolonged coronary pain in which no evidence of myocardial infarction appears The most careful observation of patients with so-called attacks of severe angina pectoris, watching for a slight fever or for significant alteration in the electrocardiograms, will be necessary to arrive at the correct diagnosis

Treatment

There is hardly any other condition in the general field of heart disease in which it is more difficult to appraise the value of specific measures of therapy than in the treatment of acute coronary thrombosis Events occur

with such suddenness that one may be too ready to attribute the last procedure employed as a cause of the result, whether favorable or unfavorable Notwithstanding this there are certain methods of treatment that for the present meet with general acceptance and others that may be regarded as still in the experimental stage or at least open to doubt

Of first importance is the relief of pain This is best obtained by the liberal use of morphine The amount necessary will vary from one dose of 0 015 gm (¼ grain) subcutaneously to many such doses When the pain is very severe and persisting morphine should be repeated in one half hour or so and at times a grain or more will be necessary Morphine should be given subcutaneously or intravenously as oral administration will be entirely inadequate Demerol in doses of 100 mg may also be tried Papaverine (0 05 to 0 1 gm given intravenously) has been suggested for severe coronary pain This may be administered every four to six hours for several days There have been isolated instances of sudden death following within one or two minutes after the intravenous use of papaverine in coronary thrombosis In fact, the intravenous injection of many drugs in cases of coronary disease occasionally causes instant fatality It does not appear to be a specific result of any one chemical compound, for the variety of drugs responsible for such sudden and unpredictable disasters is great In this group can be mentioned arsenic, mercurial diuretics, aminophylline, papaverine and caffeine The exact cause of this is obscure The abruptness of the action makes one suspect that these chemicals act as a trigger in a susceptible heart in producing ventricular fibrillation or standstill of the heart

During the early minutes after the onset, if the patient s condition seems very critical, he should not be moved or even undressed unless it is extremely inconvenient to treat him as he is These changes are often better made a few hours later When, as occasionally happens, the patient quickly becomes unconscious and pulseless the hypodermic use of adrenalin (0 5 to 1 cc of a 1 1000 solution) is indicated and may help to restore consciousness

It has been suggested that injecting a liberal amount of 0 5 per cent novocain subcutaneously in the precordial region, particularly over the painful area, may control the intractable pain of coronary thrombosis Although I have had no extensive experience with this method of treatment, it seems quite safe and may obviate the need of repeated injections of narcotics with their nauseating effects

Complete mental and physical rest is paramount The patient should be spared all possible physical movements Frequent tiring examinations are undesirable After the initial pain has subsided, sleep should be assured the first few nights, even using morphine if necessary It is just as well to avoid the use of enemas for one to several days despite the absence of bowel movements There is very little food consumed during these days and it is not necessary that the bowels should move every day An enema on the third or fourth day will often be better tolerated There is very little that can be done for the nausea and vomiting that occur during the first day or so They are partly the result of the attack and partly brought on by the

morphine that is given, generally they subside after the first day, especially if narcotics become unnecessary

Considerable dehydration develops in some of these patients They often lose a great deal of water and salt with the marked perspiration that occurs and from the vomiting and inability to retain fluids and nourishment taken by mouth It sometimes is imperative to administer 1000 cc of normal salt solution or 5 per cent glucose solution intravenously or subcutaneously Some observers have been very favorably impressed by the results of intravenous injection of 100 cc of 50 per cent glucose solution I have not witnessed any striking improvement following this procedure Another reason for encouraging the intake of fluid is the oliguria that may amount to a complete anuria so common after an attack There is also some ground to believe that the administration of sodium chloride would be desirable for the state of shock can certainly be aggravated by the great loss of salt that takes place from excessive perspiration in some cases

Oxygen may be of use When there is marked dyspnea and pulmonary congestion the inhalation of oxygen can improve breathing and cyanosis In fact I have also seen instances in which severe and persistent pain seemed to disappear promptly after placing the patient in an oxygen tent During the first hours stimulants like caffeine and adrenalin may be beneficial, the former for respiratory distress and the latter for shock and a low blood pressure It is difficult to be convinced of their ultimate favorable effects but they seem to be helpful What is much needed is some method of combating shock and the low blood pressure that is often present, for apart from other ill effects such as decreased renal flow and increasing nitrogen retention, prolonged shock probably increases the area and severity of the original myocardial infarct There is every reason to believe that plasma or albumin administered intravenously might be helpful for the profound state of shock Although my own experience is limited it seems fairly certain that in some cases this method of treatment has been lifesaving From a physiologic point of view it is conceivable that an increase in blood volume produced by plasma would be desirable at one time for shock and a decrease produced by bleeding for pulmonary edema at another time, even in the same patient In a recent case in which the pulse could not be felt, 250 cc of plasma given intravenously and repeated in four hours caused a dramatic improvement and seemed to save the patient s life Possibly some preparations such as paredrine (10 to 20 mg , intramuscularly) or ephedrine (25 to 50 mg) may prove useful In this connection it is of interest that paredrine has been shown to produce venous constriction If this is true it ought to be helpful in increasing venous return, which is much to be desired in shock

The remainder of the treatment is expectant and directed at complications Of these, two are rather important from a therapeutic point of view First is the development of complete heart block with or without Adams-Stokes syncope This is an infrequent complication but may be controlled very satisfactorily by the hypodermic injection of 0 5 to 1 cc of 1 1000 adrenalin solution In some cases it will be necessary to give such injections

frequently In several instances I have given 0 3 to 0 5 cc of adrenalin every two hours for forty-eight hours to patients who had this condition and thereby prevented the pauses of the heart that were otherwise occurring, finally observing that the tendency to syncope had disappeared It has seemed to me that in rare cases such treatment has been life saving

The other complication to be considered is paroxysmal ventricular tachycardia This occurs in about 3 per cent of the cases When it develops it can quickly produce a state of collapse The heart rate becomes very rapid (180 to 200) and the blood pressure falls still further The condition can often be recognized by three features The rapid rate, although apparently regular, may show slight irregularities which the ear can detect The first heart sound may change in quality and intensity in different heart cycles and carotid sinus or ocular pressure never produces any slowing of the heart These points can distinguish ventricular tachycardia from other forms of paroxysmal tachycardia Quinidine, procaine amide and magnesium sulfate are the drugs of choice in controlling this irregularity Digitalis will not only fail to slow it but will tend to accelerate or to perpetuate it Quinidine can stop it but the dose required varies considerably I have seen such an attack promptly stop after one dose of 0 3 gm (5 grains) taken by mouth In another case it required 1 5 gm (22 grains) to stop an attack and such a dose five times a day for several days to prevent the return of the tachycardia In two instances it was found that even very large doses of quinidine merely slowed the ventricular rate and failed to abolish the abnormal mechanism The rate would gradually return to the previously high level as the effect of the drug wore off In these two cases the hypodermic injection of 1 0 to 2 0 mg of atropine sulfate, one hour after a large dose of quinidine had been given orally (while the temporary slowing had taken place), promptly eliminated the ventricular tachycardia Quinidine may be also given intramuscularly or intravenously It would appear that the parenteral method, especially the intravenous, would carry more risk A solution of 5 per cent glucose or normal saline is made up so that 200 cc contains 1 2 gm of quinidine dihydrochloride or lactate It is given intravenously at the rate of 0 1 gm per five minutes It is helpful to be taking electrocardiograms during the injections so that treatment can be stopped the moment the desired result is obtained Quinidine merely restores the normal rhythm of the heart and controls this complication It does not prevent the other complications to which the same patient is still subject Similarly 2 0 to 4 0 gm of magnesium sulfate, given intravenously, may stop these attacks (see Chap 13)

There is a final use of quinidine that bears mention Evidence has been advanced of both a clinical and experimental nature that quinidine sulfate tends to inhibit the development of ventricular fibrillation It is more than likely that some of the instances of sudden death in coronary thrombosis are due to ventricular fibrillation I formerly advised giving 3 grains of quinidine three times a day during the first two or three weeks following an acute attack, except when there is any contraindication to its use One would want to give the smallest dose that is effective because the drug can

have undesirable effects A dose of 0 3 gm (5 grains) may be preferable for I saw one patient who was taking 0 2 gm at four hour intervals About one hour after the second dose he died instantly and the autopsy showed acute coronary thrombosis without rupture of the ventricle This is just the type of sudden death which quinidine is meant to prevent In fact, I had another experience identical with the above in which sudden death not due to rupture of the ventricle, occurred while the patient was taking 0 2 gm of quinidine three times daily The question arises whether a larger dose would have been effective The main contraindication is any evidence of defects in conduction, like bundle branch block or partial or complete auriculoventricular block Inasmuch as quinidine can further impair the conduction apparatus it should be given with caution under such circumstances It is difficult to prove that the routine use of quinidine as has been suggested has any practical merit or that the dose is adequate to produce the effect for which it is given Only much more extensive experience can answer these questions

It is the custom of some physicians to give preparations like amino phylline routinely during attacks Although there is some experimental evidence to show that such a drug increases coronary flow and that it diminishes the extent of myocardial infarction after coronary arteries are ligated, it is difficult to be certain that it is useful in clinical cases of acute thrombosis This subject, however, deserves further study Such a drug has been used even intravenously during the height of pain I have had some experience with this but it both requires and deserves further controlled observation in clinical cases to establish the usefulness of such medication

In addition to some of the things the physician is called upon to do there are certain procedures he should not do When a syphilitic patient has an acute coronary thrombosis it is inadvisable to give any intravenous antiluetic treatment In fact it is better to disregard the syphilitic aspects of the problem entirely during the first two weeks or so In these cases it is not at all certain that syphilis is directly related to the coronary thrombosis for it is more likely that the latter is due to the same causes that are at work in nonsyphilitics Reference has been made previously to the use of insulin in cases of coronary thrombosis This should be avoided as much as possible *because for an hour or two after its use the work of the heart is increased* about 20 per cent if the blood sugar drops too low, and this added strain may be very serious It is well known that attacks of angina can be precipitated by insulin and I have seen a rupture of the ventricular wall occur about one-half hour after 10 units were given Insulin may be given cautiously if the blood sugar is high and marked fall of the blood sugar can be avoided

The same question of insulin administration in other cardiac disorders deserves a word of comment I have frequently seen patients with various types of heart disease (congestive failure or angina pectoris) improve strikingly on decreasing the dose of insulin or discontinuing its use entirely It would appear that such patients were treating the diabetic state too thoroughly, having a blue urine test quite constantly One wonders whether it

would not be desirable for cardiac patients to have a slight glycosuria The sugar in the urine may act as a diuretic In one case attacks of recurrent acute pulmonary edema ceased on omitting insulin and permitting the urine to contain 2 per cent sugar The attacks in this case used to come some hours after the insulin injection, at a time when hypoglycemia may have been present

If a patient has an acute coronary thrombosis and some other surgical condition, such as an obstructing prostate or an infected kidney that needs removal, it is advisable to delay operation for at least a month or two until a satisfactory recovery of the heart has taken place There is a final precaution in the care of patients with coronary artery disease It has been observed that an attack of coronary thrombosis can occur directly after the intravenous injection of the iodine dye that is used for cholecystograms I have seen two patients with angina pectoris in whom an attack of coronary thrombosis was brought on a few minutes after the intravenous injection of the dye It is wiser, therefore, when a patient is either known to have or suspected of having coronary artery disease and it is desired to study the condition of the gallbladder, that x-ray examination should be made only after oral administration of the dye

Finally, the question arises whether or not digitalis should be employed in acute coronary thrombosis There are many theoretical reasons one could give for or against its usefulness I have always felt that it is more likely to do harm than good If persistent auricular fibrillation is present it should be given but this condition is very rare, as when it occurs during an attack it is almost always transient Furthermore, when there is peripheral pitting edema, engorged liver or hydrothorax, digitalis should be administered as in any case of congestive heart failure This is rare, however, except after the first few weeks It is uncertain whether or not it is beneficial in those cases showing rales in the lungs and dyspnea The drug should be helpful for left ventricular failure but might be harmful for the peripheral failure or shock, and both conditions exist simultaneously in the early days of acute coronary thrombosis The lack of cardiac dilatation would make one hesitate in using the drug Although much more statistical evidence will be required before the question can be finally settled, at present it seems best not to use digitalis routinely during the first two weeks of acute coronary thrombosis

The general nursing care of the patient is very important Everything should be done to make him comfortable The diet during the early days should be confined to liquids, gradually returning to more ordinary food in small amounts Many of these patients are overweight and to obtain a loss of weight during this illness is desirable It has been advised that a low caloric diet (500 to 800 calories a day) should be used in acute coronary thrombosis and in fact is being advocated in the treatment of any severe or stubborn case of congestive heart failure (Proger and Master) It has been found that this semistarvation diet diminishes the work of the heart and produces other favorable effects on the circulatory dynamics Patients should be kept at rest for four to eight weeks Occasionally the period of

rest or restricted activities needs to be longer, but sometimes three weeks appear to be sufficient From a purely clinical point of view the four to eight week period has been found to be adequate and it is of interest that in large animals it has been observed that two to five weeks are required to establish adequate collateral circulation after experimental ligation of a major coronary artery It is a general biologic principle that the main stimulus for the development of collateral arterial circulation is a local need for it Inasmuch as the present evidence favors the view that there are anastomoses from one main coronary artery to another normally, even in infancy, it is not difficult to conceive of the mechanism of recovery from myocardial infarction Economic and social factors often will determine the exact length of time of convalescence Whether nurses will have to be employed is also often a matter of the financial status of the patient It generally is advisable that the patient should not be permitted to go to the bathroom for either bowel movement or urination during the early days Often it proves to be less of a hardship to permit the use of a commode than that of a bed pan These details need to be worked out by the physician in individual cases After the period of bed care the patient should spend a week or two in the process of getting out of bed sitting up in a chair for a short while, then gradually increasing the period each day There is a growing tendency to permit many of these patients to be treated out of bed, in a chair I am inclined to endorse this view in many cases and feel ready to permit greater activity of the legs in all cases than was the custom formerly When the patient becomes ambulatory, return to activities should be gradual and in most cases it will be desirable to urge that he should permanently diminish his activities to some extent

The above program of rest is the one generally accepted by many physicians at the present moment During the past year or so I have been treating an increasingly large proportion of cases by having them spend the greater part of the day out of bed in a chair, even from the onset of the illness Just as soon as the initial stage of shock and severe pain has passed, the patient is allowed to sit in a comfortable chair with his feet down for as much of the day as he likes He is helped back to bed to sleep and is not allowed to walk or permitted any other exertion during the first two weeks or so It is too early to offer statistical evidence as to the validity of this method of treatment, but so far it seems to be very promising and I suspect that it will actually decrease both the morbidity and the mortality of this illness

New methods of therapy will necessarily be proposed in the future Attempts will be made to prevent further thrombosis of vessels Mural thromboses of the ventricular cavities with subsequent arterial emboli are the cause of some fatalities One not infrequently sees patients survive the first week of the attack then while everything seems to be progressing satisfactorily a sudden cerebral embolus occurs with hemiplegia that proves fatal In such cases if the secondary mural thrombus in the left ventricle could have been prevented from forming during the first several days after the onset of the attack fatalities would not have occurred These thrombi

are found in about one third to one fourth of the patients dying in the acute attack and therefore form a considerable group

Another frequent complication is thrombophlebitis and secondary pulmonary embolism Emboli of one type or another are a not uncommon cause of death in cases of coronary thrombosis Primarily for these reasons anticoagulant therapy has now come into general practice At the time of the last edition of this book (1945) I had begun a study of this problem but the work was interrupted by World War II before sufficient experience could be obtained Now, rather extensive studies by Wright and Nichols have shown that the mortality from acute coronary thrombosis under anticoagulant therapy using Dicumarol dropped from 23 per cent in the control group to 16 per cent in those treated, and the incidence of thrombo embolic episodes from 19 to 9 per cent

From the above figures one is led to conclude that anticoagulant therapy is valuable for acute coronary thrombosis There are several possible beneficial effects of its use The first is to prevent thrombophlebitis of the legs and subsequent pulmonary emboli, a serious and frequent complication There is very little doubt but that Dicumarol properly administered can prevent or markedly diminish the incidence of this complication The second purpose is to prevent the development of a ventricular mural thrombus that is so common, with the hazard of arterial emboli, especially to the brain It is not so certain how effective Dicumarol therapy is in this regard Inasmuch as these thrombi are known to form the first few days (while the thromboses in the veins of the legs occur much later), therapy might have to be effective very early and promptly The third and more doubtful purpose is to affect favorably the actual thrombus in the coronary artery or to prevent secondary and further coronary thromboses

There are several courses of anticoagulant therapy one might undertake One is to use heparin, the second is to use Dicumarol and the third is to combine the two drugs For the latter purpose Dicumarol would be started and heparin would also be given only the first thirty six to forty-eight hours (100 mg intravenously every six hours) until the effect of the former drug became manifest All these measures are difficult, expensive and can hardly be carried out unless the patient is in a hospital Satisfactory results comparable to those obtained with Dicumarol have been obtained by giving heparin/Pitkin menstruum, 300 to 400 mg every second day intramuscularly With this method the coagulation time of the blood needs to be frequently checked, trying to keep it at 30 to 45 minutes or about three times normal If the effect of heparin is excessive or bleeding occurs, the antidote is protamine, about 50 mg intravenously This checks the bleeding tendency in a few minutes The method most commonly employed, however, is to administer Dicumarol orally

At the outset it must be realized that there are certain contraindications to anticoagulant therapy These are primarily severe renal or hepatic insufficiency, jaundice, purpura or severe blood dyscrasias, postoperatively in brain or spinal cord cases, immediately after any major operation, open

wounds and severe nutritional deficiency These conditions rarely interfere in the treatment of coronary thrombosis Before treatment is started the determination of the prothrombin time is performed If this is in the vicinity of 100 per cent of normal, 300 mg of Dicumarol is given If it is definitely lower the initial dose is appropriately diminished The prothrombin time is then tested every morning and the subsequent dose regulated accordingly The aim is to keep it around 20 per cent of normal or about 35 seconds The second dose frequently will be 200 mg and the daily dose thereafter 100 or 50 mg It may be necessary to omit the Dicumarol entirely for one or more days if the prothrombin time is excessively prolonged It certainly should be stopped if the time is above 40 seconds The treatment is continued for three, four or more weeks, preferably until the patient is at least partially ambulatory or until two or three weeks have elapsed after the last thrombo embolic episode has occurred

During this treatment careful watch for evidence of bleeding should be carried out, particularly from the nose in the urine or in the stools If significant bleeding does develop or if the prothrombin time is very excessive vitamin K_1 in 50 to 100 mg doses intravenously should be given If that is not effective whole blood transfusions should be used It has been found *that certain commercial preparations like hykinone are ineffective* for this purpose If this program is carried out with care, serious bleeding will be very rare It is obvious that this treatment is difficult and expensive It would help greatly if some quick and reliable bedside test for prothrom bin activity could be devised so that Dicumarol could be given in patients homes On occasions I have carried out this course of treatment at home but it required a good deal of cooperation At present it appears that anticoagulant therapy has definitely decreased the mortality of acute coronary thromboses, mainly by diminishing the incidence of thrombo embolic complications and should be started as soon as the diagnosis can be made if there are no contraindications In fact, anticoagulants ought to be valu able as a preventive in those cases that present the premonitory symptoms which occasionally appear a few days before the acute dramatic episode

One of the most common and harmful errors in the management of acute coronary thrombosis is to outline a lengthy period of invalidism and con valescence There are too many patients who are told to stay in bed three to six months or to spend a year away from work taking it easy When there is congestive failure there may be no other choice, but in the absence of congestion, even if chest pain continues, such long invalidism is not only fruitless but often harmful It generally will be found that the degree of pain will be no greater if the patient is ambulatory What is more aimless is to advise restricted activities, or as is frequently done, confinement in bed, merely because of weakness After the first two months, such weakness will not be helped by a program of inactivity The entire illness, coupled with the alarming precautions taken by the physician and family produces a state of fear, which often results in a profound neurasthenia and depression Considerable weakness is necessarily part of the illness

itself This is further accentuated by the immobility during the early weeks in bed To this is then added a neurasthenic state that finally may condemn the individual to a permanent useless existence

Sometimes these prolonged periods of 'convalescence' are the result of insurance considerations, for total permanent disability does not begin to take effect until an illness has lasted over three months At times one is led to believe that this type of insurance has done more harm than good for it has destroyed ambition and encouraged invalidism What is needed to help this weakness is to encourage the patient to increase his activities and to assure him that his heart has recovered sufficiently to do more If this point of view is stressed early in the illness, much unnecessary invalidism will be prevented Many patients ought to be doing part time work in two months and the majority in three to four months What has been said about prolonged bed care for patients with coronary thrombosis applies to some extent to patients with other types of chronic heart disease and to those with many other noncardiac conditions Not only is prolonged bed rest frequently unnecessary but it may be conducive to the development of further complications such as hypostatic pneumonia, prostatic obstruction, renal stone formation and phlebitis with pulmonary embolism

Finally, if a patient has had two or more attacks of coronary thrombosis and has recovered, one might be tempted to administer Dicumarol constantly This is being done by some physicians and appears to be a reasonable procedure The daily dose is likely to be about 50 mg , and careful observation will be necessary One might need to perform a prothrombin test weekly and try to keep the prothrombin time around 25 to 30 seconds This method of prolonged ambulatory Dicumarol therapy might also be applied to an occasional case of repeated arterial emboli in valvular disease

7

Hypertensive Heart Disease, Arteriosclerosis, "Chronic Myocarditis," and Rare Forms of Heart Disease

HYPERTENSIVE HEART DISEASE AND ARTERIOSCLEROSIS

Hypertension has a most important bearing on the subject of heart disease As a concomitant factor in heart failure it probably surpasses all others, so that in most general surveys, hypertensive heart disease heads the list of disabling forms of heart failure The exact role that is played by the actual elevation of the blood pressure is by no means clear, for so often the same degree of hypertension is well tolerated for many years by one patient and results in severe cardiac insufficiency in another Even the causes of hypertension are difficult to understand for it seems to accompany a variety of conditions How much can be explained on the basis of permanent structural changes within the body, especially the arteries, and how much on the basis of an altered nervous or functional state always requires consideration The marked and even sudden changes that have been observed in the level of the blood pressure in many individuals have necessarily resulted in the firm conviction that the emotions, the nervous system, the endocrine glands and the vasomotor apparatus are all intimately related to this problem

At the outset it must be recalled that there are a few distinct clinical conditions which, although not really related to the general problem of essential hypertension produce or are accompanied by some elevation in blood pressure It is of some importance to be familiar with these conditions for in so far as they exist, physical examination will reveal certain findings that will explain why these particular individuals are hypertensive In coarctation of the aorta (Chap 11), for example, there is hypertension in the arms although the pressure in the legs is low Here the elevation in the blood pressure that is found on routine examination is directly due to a structural abnormality Similarly one frequently finds hypertension in patients with prostatic obstruction In some, the pressure will remain elevated until the obstruction is relieved e g by an indwelling catheter, and then it will fall considerably Such a fall of the pressure has been observed when all other factors, such as rest in bed, have been adequately controlled At times this fall may be excessive and it will then be necessary to temporize and delay the prospective prostatectomy until a partial recovery of

the blood pressure has taken place. There are many instances in which the eventual prostatectomy produces a permanent lowering of the blood pressure.

Other conditions that are associated with and in some way productive of hypertension are polycystic kidneys, eclampsia, tumors of the adrenals, pituitary basophilism, occasional instances of sudden intracranial hemorrhage, acute nephritis and polycythemia. Hyperthyroidism is also commonly associated with hypertension but the level of the blood pressure falls little if any after the basal metabolism is brought to normal by operation. In addition, with certain cardiac disorders there is an elevation of the pressure which is the result rather than the cause of the disturbance in the heart. When complete heart block develops, the systolic pressure is apt to rise and the diastolic to fall purely as a result of the very slow heart rate. Somewhat similar changes follow the development of free aortic regurgitation. Finally, when hypertension accompanies congestive heart failure, more often than not the pressure level falls as improvement takes place on rest in bed and digitalis. After a considerable experience I feel convinced that some of the fall in blood pressure is a direct result of cardiac therapy and disappearance of dyspnea and congestion and not to be accounted for on the basis of psychic influences or rest in bed. In fact, if the blood pressure *does not fall while treating a patient with hypertensive heart failure,* it generally indicates that improvement in the heart is not taking place or will not be so great as when the pressure does fall. This relationship is mentioned because there still prevails the idea that when the heart improves it manifests this improvement by an increase in blood pressure. Although *this is often the case if the blood pressure is very low to begin with, it is* not the general rule when congestive failure takes place with hypertension.

There is one more peculiarity of the blood pressure in relation to heart disease that merits consideration. There is a group of patients having hypertension and angina pectoris in which, following an attack of coronary thrombosis, the blood pressure falls with a complete or partial disappearance of the anginal attacks. When such patients become ambulatory some will thereafter have a low blood pressure, despite the fact that their general health remains even better than before the attack of coronary thrombosis, as they no longer have anginal spells. Under these circumstances a reduction in the blood pressure has taken place following an injury to the heart. This cannot be explained on the basis of structural alterations in the peripheral vascular tree. It seems more reasonable to assume that the previous elevation in the blood pressure was caused reflexly from an irritable focus in the heart and after the coronary thrombosis, the local cause was removed. These experiences and those cited in the previous paragraph in which it seems that digitalis may diminish the blood pressure by improving the heart, make one suspect that apart from all other causes hypertension in some cases may have its origin reflexly in the heart.

Apart from the conditions just mentioned hypertension is regarded as an accompaniment of chronic nephritis or pyelonephritis, or as an idio-

pathic or essential hypertension The latter type is most common One suspects that to a large extent or at least in its early stages it is functional or neurogenic and not based on irreversible structural disease One cannot explain the oscillations that the blood pressure level undergoes in some individuals on the basis of organic disease I recall seeing a woman 55 years old whose pressure was known to be over 200 mm for some years After three weeks rest in bed there was a steady gradual decline to 140 mm from the initial level of 240 mm Thirty minutes after reading a letter containing very distressing news the level immediately jumped 100 mm back to the original figures of 240 mm Such changes cannot be explained except as a result of nervous or humoral influences

The experimental work of Goldblatt has shown that the production of renal ischemia by constricting the renal artery causes maintained hypertension in animals It has also been shown that even after such hypertension has lasted many months removal of the ischemic kidney restores the blood pressure to normal Furthermore, this type of hypertension was found to be independent of the sympathetic nervous system The inference from this work is that some product is liberated from the abnormal kidney which circulates in the blood and causes hypertension This concept appears to be of great promise affords a clearer understanding of the entire question of hypertension, and may possibly lead to practical therapeutic advances In fact already in a few cases of unilateral nephrectomy for hypertension and chronic pyelonephritis the blood pressure has returned to normal

Etiologic Factors

Among the few known etiologic factors the most important is heredity There are families with strong tendencies to hypertension and others which tend to hypotension It has impressed me that marked freckling of the back of the forearms is unusually common in hypertensives and probably denotes a vascular vulnerability The young nervous hypertensive or potential hypertensive often has a slight fever (99 6 F) The females predominate over the males in the proportion of about three to two The menopause seems to be a common period in life in which these changes become prominent although earlier evidences of this tendency are generally available The hypertensive person will often manifest certain stigmata of neurovascular vulnerability in earlier life Nosebleeds menstrual disturbances flushing—especially of the neck—migraine palpitation and nervousness will frequently b ound Furthermore, it will be noted that they often have a tendency to slight elevation in the blood pressure on emotional provocation years before they become permanenetly hypertensive An insurance examination or a visit to a consultant may raise the pressure to a higher level than the one found by the patient s family physician In fact, the one who is not prone to hypertension is not apt to show this rise even on nervous tension It seems therefore that the pressure is unusually labile in these vulnerable individuals and that they have transient hyper-

tension for years before they develop what one might call permanent hypertension

Although hypertension and arteriosclerosis are frequently associated, it is unlikely that the latter is the cause of the former One may see extensive sclerosis of the large arteries without hypertension and very little arteriosclerosis with marked hypertension It is more plausible that prolonged hypertension eventually leads to sclerosis of the arteries, but that during the early years spasm of vessels otherwise sound is going on The fact that the first portion of the pulmonary artery is rarely sclerosed except in cases of mitral stenosis or other conditions in which pulmonary pressure is elevated, lends support to the role hypertension may play in producing arteriosclerosis When the blood pressure has been elevated for any considerable length of time, although the radial and brachial arteries may not show much evidence of sclerosis changes in the retinal arteries will almost always be present Retinal arteriosclerosis can be judged from the irregularity of the caliber of the arteries on ophthalmoscopic examination and the nicking of the veins at the points where they are crossed by the arteries When the process is more advanced, hemorrhages, exudate and papilledema may be found

There are a few diseases that, in so far as they may affect blood vessels, may eventually predispose to early hypertension, e g , typhoid fever, syphilis, chronic lead poisoning, gout and rheumatic fever There is some suspicion that this last disease may be a more frequent precursor of hypertension than has been supposed There is no evidence that foci of infection in teeth or tonsils are of any importance in this question In the great majority of cases of hypertension, however, none of these infections has been responsible and for the present at least they are best regarded as 'essential or idiopathic

Effect of Arteriosclerosis on the Heart

Before discussing hypertensive heart disease the possible effect of arteriosclerosis on the heart must be taken up The term arteriosclerotic heart disease has led to much confusion In the minds of some this term signifies heart failure resulting from peripheral sclerosis and in others it means coronary arteriosclerosis If it merely includes those cases in which there is arteriosclerosis of the coronary arteries then it should be called by its proper name, i e , coronary artery disease If the former idea is held then it is a misnomer, for there is little if any evidence to show that sclerosis of the peripheral arteries has any appreciable effect on the efficiency of the heart A study carried out at the Peter Bent Brigham Hospital analyzing all the postmortem material in which there was a high degree of peripheral arteriosclerosis showed that when those cases were omitted in which there was hypertension, significant coronary artery disease or other obvious causes of cardiac disability, such as valvular disease in the remainder there was neither clinical nor pathologic evidence of heart disease The death of these patients was due to some surgical or

noncardiac cause In a word, it was found that even an extreme degree of peripheral arteriosclerosis had no deleterious effect on the heart It is suggested, therefore, that the term arteriosclerotic heart disease' be given up

Blood Pressure Determination

A word about the actual taking of the blood pressure seems worth mentioning After the patient has relaxed, with the arm in a comfortable position, the pressure is increased above the expected systolic level The mercury or pressure should be permitted to fall slowly while auscultating below the cuff Many physicians do this too rapidly and inaccuracies of 20 mm or more may result Furthermore, it is important that the first time a patient is examined, palpation of the radial pulse should be performed to check the auscultatory determination If this is not done one may occasionally start the examination with the pressure level at the auscultatory gap at which point no sounds are heard and yet the true reading may be 40 to 60 mm higher It is obvious that such an error can easily be avoided, for the radial pulse would be palpable even in this silent zone and the observer would have had to increase the pressure and then would have found that sounds return Although the auscultatory method is more satisfactory than the palpatory, the latter at times will obviate mistakes I recall a case in which the physician remarked that he could not obtain the blood pressure It was found that the diastolic blood pressure in this young girl was 180 mm and the physician always started his readings at about 150 to 160 mm If he had felt of the radial pulse he would have known that the blood pressure must have been higher, for the pulse had not been obliterated Finally, there are instances in which a violent or hyperdynamic pulse, such as is seen in aortic regurgitation, produces a shock and noise with each beat as it strikes the cuff and this is audible in the antecubital space It may give a falsely elevated systolic blood pressure In such a case palpation of the radial pulse will indicate the systolic level I have seen an instance of this type where the actual systolic reading was 140 mm when it had been read as 250 mm *In this case a definite sound could be heard with the pressure over 300* mm but it was an impact transmitted from the top of the cuff

Clinical Course

In following the clinical course of a patient with hypertension there are several definite things for the physician to bear in mind The exact level of the blood pressure is by no means the important criterion of the patient s progress The pressure or the associated arterial disease will produce clinical disease in one of several ways Of greatest importance is what is going on in the heart This effect may become manifest in one of two ways Angina pectoris or coronary thrombosis may result from involvement of the coronary arteries or there may develop congestive heart failure, especially left ventricular failure with paroxysmal dyspnea

Next in importance is the possibility of cerebral hemorrhage There is no method that enables one to predict which hypertensive patient will develop a cerebral hemorrhage or when it may be expected The current opinion is that those with high diastolic pressures are most prone to cerebral accidents I have had the suspicion that the reverse i true It has seemed that most patients who develop a sudden hemiplegia have had pressure levels of about 200 to 240 mm systolic and 100 to 120 mm diastolic Those with diastolic readings of 140 to 160 mm are more apt to have general encephalopathy, cardiac or renal failure but not an outspoken cerebral hemorrhage May not the high pulse pressure be the more important factor in causing rupture of a large cerebral vessel?

Another group will begin to show significant disease of the kidneys and present the picture of chronic vascular nephritis, eventually developing uremia A smaller number will have disabilities due to arterial disease of the legs, suffer intermittent claudication or even arteriosclerotic gangrene Finally there are some in whom the arteriosclerosis of the abdominal vessels is productive of symptoms Many ill-defined complaints, i e, indigestion and abdominal pain, are no doubt due to arteriosclerosis of the mesenteric vessels In this same connection the mild diabetes that is so common in elderly hypertensives may well be due to arteriosclerotic changes in the pancreatic vessels Frequently the same patient with hypertension may show several of these evidences of arteriosclerosis He may have a mild glycosuria, slight diminution in renal function and definite intermittent claudication Another may have outspoken angina pectoris, no renal impairment and more marked sclerosis of the arteries of the legs Finally, extensive arteriosclerosis may be present without hypertension It is the function of the physician to try to estimate which *is the major route that the vascular degeneration is taking, and in general* it will be found that the cardiac complications are the most important

Exactly when the heart will begin to fail in an individual with hypertension is a very variable matter Neither the level nor the duration of the elevation in the blood pressure can entirely account for the development of cardiac insufficiency In many instances it is clear that the main determining factor is the integrity of the coronary arteries Atheromatous changes and narrowing of these vessels can account for the development of cardiac embarrassment in some individuals with only a moderate hypertension But there are other instances in which the gross appearance of the coronary arteries is essentially normal and yet the heart muscle fails This unknown factor accounts for much that determines the whole question of congestive heart failure I suspect that this factor "X" is linked up with the finer vascular bed of the coronary system It is well known that throughout the human body there are a great many more capillaries than are being used under ordinary circumstances A skeletal muscle or a glomerulus of the kidneys contains ten times as many capillaries as are functioning at any particular time The others are closed and are resting for the moment Under special circumstances the number increases, the

walls are opened and blood corpuscles begin to flow through This constitutes the reserve function of the organ, and may not cardiac reserve depend on this very ability to open up new capillaries? When the heart muscle fails and yet shows no obstruction in the main coronary arteries may not the cause lie in some structural or functional defect that prevents the opening of these terminal reserve channels? It is conceivable that if these abnormalities do exist, they might yet be overlooked by the methods of pathologic study customarily employed in postmortem examination

In the course of time an individual with hypertension may develop cardiac failure In some this may not occur until twenty years have elapsed, in others the downhill path begins after a short time The heart generally becomes enlarged but the degree of hypertrophy varies considerably in different cases and is not directly proportional to the height or duration of the hypertension What concerns us here is the development of congestive heart failure and not of angina pectoris The latter condition can appear any time and suddenly alter the clinical course that the particular case might otherwise have taken Most commonly, in the former, the first symptom is breathlessness This appears at the outset on a degree of effort that formerly was well tolerated Dyspnea on walking is, therefore, the most frequent early evidence of myocardial insufficiency In many cases unwonted fatigue may have preceded this but because of the numerous noncardiac causes of fatigue it does not serve so well to identify the condition as heart failure There is a considerable group of hypertensives in which the shortness of breath appears suddenly, especially at night and may even take the form of acute pulmonary edema The term cardiac asthma is often used to classify these cases The term is objectionable because the word asthma has too closely associated with it a bronchial condition that has nothing to do with the heart and is comparatively benign It is better to designate the condition by such terms as paroxysmal cardiac dyspnea nocturnal dyspnea or acute pulmonary edema as the circumstances warrant

When dyspnea develops, either of the gradual or of the acute form, it indicates failure of the left ventricle Examination often shows cardiac enlargement in addition to the elevation in blood pressure The rhythm of the heart may be perfectly regular or arrhythmias due to extrasystoles or auricular fibrillation may be present When the auricles are not fibrillating a gallop rhythm and pulsus alternans are frequently present These two signs are so common and so important in hypertensive heart disease that they should always be looked for The former is detected by careful auscultation over the precordium where the sounds have a peculiar quality resembling a canter due to an extra third heart sound in diastole The latter is detected by palpation of the radial artery or better still on determining the blood pressure While auscultation is carried out below the blood pressure cuff, just as the first sounds become audible, the pressure should be prevented from falling for a moment and the sounds will be heard to alternate in intensity This method is even more helpful

than palpating the radial artery Occasionally the same mechanism of alternation of the ventricles will produce an alternating intensity of the heart sounds, murmurs or the apex impulse over the precordium Similarly the gallop rhythm may result in a bifid apex impulse that can be seen and felt

Gallop Rhythm

It is appropriate at this point to discuss the clinical significance of gallop rhythm This auscultatory finding is most important, can easily be detected but is often overlooked The extra sound is generally heard in *diastole, i e, between the second and first heart sounds at the apex (Fig* 185) When the extra sound follows the second sound it is called protodiastolic, when it just precedes the first sound it is presystolic and when it occurs in mid-diastole it is called mesodiastolic Such terminology is difficult and unnecessary from a clinical point of view Gallop rhythm occurs most commonly in hypertensive heart disease and coronary artery disease and less frequently in rheumatic valvular disease Occasionally it is present during acute infections such as diphtheria and rheumatic carditis Because it is rarely associated with auricular fibrillation, the contraction of the auricles must have something to do with the production of the gallop In fact, I have seen instances in which a typical diastolic gallop disappeared with the onset of auricular fibrillation and reappeared with the return of regular rhythm In dogs, gallop rhythm has been found to be related to increased pressure in the left auricle In the nonvalvular cases in which gallop rhythm is manifest bundle branch block is also often present *It almost always denotes a fairly serious affection of the heart* muscle, carrying with it a life expectancy of one to three years, although occasionally such patients do live longer I have seen exceptional instances in which a true diastolic gallop rhythm was detected on several different examinations (once during pregnancy) in the absence of any other evidence of heart disease and finally disappeared, the patients remaining free of any organic heart disease The third sound of a gallop rhythm must be distinguished from a normal sound heard in mid diastole in some healthy hearts This is not generally difficult for the rate is slow in the latter and somewhat accelerated in the former Furthermore, patients with a normal third heart sound are apt to be young and show no other evidence of heart disease Another confusion may arise when a patient who is developing mitral stenosis first shows a third sound in diastole (opening snap) This may resemble a gallop and subsequently it will be found that the extra sound in fact was the early manifestation of what later proved to be the mid-diastolic or presystolic murmur of mitral stenosis A gallop rhythm in a patient with rheumatic carditis may be indicative of delayed conduction of impulses from auricles to ventricles (increased P-R interval) This finding may enable the examiner to predict that there is a conduction defect The prognosis of the gallop need not be grave when it is present in young rheumatic cases Even the ordinary gallop may disappear when the clinical condition improves, especially when it occurs in con

junction with acute coronary thrombosis or if the heart rate slows, for it is rarely present when the rate is around seventy

Finally a word must be mentioned about another type of gallop rhythm When the extra sound lies between the first and second heart sounds it is called a normal midsystolic gallop (Fig 184) It can be distinguished from the serious diastolic gallop by the following procedure If the stetho scope is moved from the apex to the base of the heart in rhythm with the heart beat it will be found that the extra sound, which occurs in the middle of the three sounds, gradually diminishes in intensity and finally disappears, leaving the two normal first and second heart sounds The important point is that the midsystolic gallop rhythm is benign and the diastolic is grave

Cardiac Murmurs

The finding of cardiac murmurs in patients with hypertensive heart disease is inconstant In many of these patients there are no murmurs More often a systolic murmur will be heard at the apex or base of the heart This can be either faint or fairly loud When there is no additional organic valvular lesion the louder apical systolic murmurs probably result from a relative mitral or tricuspid insufficiency and the aortic systolic murmurs from dilatation of the aorta The dilatation of the ventricular cavities which sometimes is very marked can readily account for stretching of the rings at the base of the valves even when the leaflets themselves are not particularly diseased Occasionally even the aortic valve may thereby become incompetent in hypertension and result in the presence of an aortic diastolic murmur Inasmuch as valvular lesions due to other causes, such as rheumatism or syphilis may coexist with hypertension the corresponding murmurs from these valvular defects may be found

There is one unusual development in hypertensive cases that is called Bernheim s syndrome This may also occur in other instances of left ventricular hypertrophy and strain It consists of the clinical evidence of right sided heart failure without pulmonary congestion due to displacement of the interventricular septum to the right, resulting from a large left ventricle with increased left intraventricular pressure This produces obstruction through the right ventricle somewhat analogous to what occurs in tricuspid stenosis There is increased venous pressure and there may be a normal circulation time The right auricle may also be enlarged

Involvement of Lungs

The lungs often show evidence of stasis In the milder cases a few moist rales will be found at the bases of the lungs When the degree of heart failure is more marked free fluid in the pleural cavities will develop In the fulminating cases of acute left ventricular failure a generalized pulmonary edema quickly appears with moist bubbling rales throughout the chest, cough and frothy pink sputum Such a dramatic storm may come and disappear in an hour or two With the embarrassed respiration, Cheyne-Stokes breathing is frequently present This may not be detected

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Gallop Rhythm

It is appropriate at this point to discuss the clinical significance of gallop rhythm This auscultatory finding is most important, can easily be detected but is often overlooked The extra sound is generally heard in diastole, i e, between the second and first heart sounds at the apex (Fig 185) When the extra sound follows the second sound it is called protodiastolic, when it just precedes the first sound it is presystolic and when it occurs in mid-diastole it is called mesodiastolic Such terminology is difficult and unnecessary from a clinical point of view Gallop rhythm occurs most commonly in hypertensive heart disease and coronary artery disease and less frequently in rheumatic valvular disease Occasionally it is present during acute infections such as diphtheria and rheumatic carditis Because it is rarely associated with auricular fibrillation, the contraction of the auricles must have something to do with the production of the gallop In fact, I have seen instances in which a typical diastolic gallop disappeared with the onset of auricular fibrillation and reappeared with the return of regular rhythm In dogs, gallop rhythm has been found to be related to increased pressure in the left auricle In the nonvalvular cases in which gallop rhythm is manifest bundle branch block is also often present It almost always denotes a fairly serious affection of the heart muscle, carrying with it a life expectancy of one to three years, although occasionally such patients do live longer I have seen exceptional instances in which a true diastolic gallop rhythm was detected on several different examinations (once during pregnancy) in the absence of any other evidence of heart disease and finally disappeared the patients remaining free of any organic heart disease The third sound of a gallop rhythm must be distinguished from a normal sound heard in mid diastole in some healthy hearts This is not generally difficult for the rate is slow in the latter and somewhat accelerated in the former Furthermore, patients with a normal third heart sound are apt to be young and show no other evidence of heart disease Another confusion may arise when a patient who is developing mitral stenosis first shows a third sound in diastole (opening snap) This may resemble a gallop and subsequently it will be found that the extra sound in fact was the early manifestation of what later proved to be the mid-diastolic or presystolic murmur of mitral stenosis A gallop rhythm in a patient with rheumatic carditis may be indicative of delayed conduction of impulses from auricles to ventricles (increased P-R interval) This finding may enable the examiner to predict that there is a conduction defect The prognosis of the gallop need not be grave when it is present in young rheumatic cases Even the ordinary gallop may disappear when the clinical condition improves, especially when it occurs in con

an advanced and rapidly developing form of the same process It is associated with a very high diastolic pressure the readings often being 250 systolic and 150 diastolic The course is a rapid downhill one, ending fatally in about a year or two Such patients complain a great deal of headache, may have convulsions and show marked changes on ophthalmoscopic examination including papilledema These cerebral features are described by the term hypertensive encephalopathy and may be confused with tumor of the brain This disease is not rare in younger individuals under 40 and even in those in the twenties, and is generally refractory to treatment It has been of interest to me that whereas essential hypertension in general is much more common in females, malignant hypertension is more common in males Do the endocrine glands determine this difference?

A pyrogen method of treatment for malignant hypertension has been recommended by Page Although various methods of inducing fever may produce somewhat similar results he recommends a soluble bacterial pyrogen called pyromen A dose of 0 5 cc (containing 50 gamma of solid per 1 0 cc) is given intravenously five to six days a week The dose is steadily increased as tolerance increases in order to obtain a temperature of 103 to 104 F The treatment is continued for several weeks and even a few months Clinical improvement and a fall of blood pressure is generally observed in one to two months If the treatment is ineffective or not well tolerated it should be omitted for three to seven days and then reinstituted, starting with about one half the last dose Should the febrile reaction itself become distressing aspirin or aminopyrine can be used to counteract the effect Although I have had no personal experience with this method of treatment the ordinary hopelessness of malignant hypertension warrants the trial of even such lengthy and troublesome procedures

The surgical treatment for hypertension is now in the process of being investigated Many hundreds of patients have already been operated upon, some type of dorsolumbar sympathectomy having been utilized The operation devised by Smithwick appears to have certain advantages and has gained some support It is still too early to arrive at a final judgment, but at present it appears to be successful in an appreciable number of cases and can be recommended in selected individuals It would be well, however, to perform a routine pyelogram examination in all cases of hypertension, if a complete study is desired, for occasionally unilateral renal disease may be detected by that method only On rare occasions a unilateral pyelonephritis or aberrant renal artery or adrenal tumor will be unexpectedly found It must be appreciated that tumors of the adrenal not only cause paroxysmal hypertension but also permanent hypertension The importance of these unilateral lesions is that, although rare, they may be curable by surgical means

In the selection of patients for dorsolumbar sympathectomy there are various factors to be considered The results are likely to be better in the female than in the male, if the diastolic pressure is over 100 mm and the

pulse pressure is small, in cases of pyelonephritis than in others with the same functional state of the kidneys, and in patients under 50 years of age than in older individuals The response to the cold pressor test is more valuable than the response to sedatives

Because the clinical course of uncomplicated essential hypertension is so variable and unpredictable and so many may live in essentially good health for two decades or more, it will be a long time before the evidence will be convincing that surgery has been worthwhile However, when operations are performed on those hypertensive patients who already show grave cardiac involvement such as angina pectoris, coronary thrombosis, congestive failure, paroxysmal dyspnea or gallop rhythm, it will not be necessary to wait ten or more years or to accumulate thousands of cases for statistical proof that the course of the illness has been altered Even if a smaller number of cases with these cardiac complications are operated upon and found to be well, improved or living five years after operation we will be fairly certain of the beneficial effects of surgery I have already seen a small number of such cases markedly improved and return to a useful occupation when it did not appear possible that any such recovery would have occurred on medical management At present, therefore, I am more interested in advising sympathectomy in those hypertensive patients who already have serious cardiac complications (provided kidney function is satisfactory) than as a prophylactic measure in those who are still in fairly good general health

Several years ago Kempner introduced the rice diet as a treatment for hypertension This has caused considerable controversy among many physicians This diet which consists only of rice and fruit is extremely low in sodium, containing less than 500 or 250 mg daily It is also very low in protein, fat, total calories and vitamins Patients therefore lose some weight and require supplementary vitamins There is no doubt that this rigid diet lowers the blood pressure in a large number of cases What is disputed is whether the effect is entirely due to the low salt intake Most investigators believe that the beneficial effects could be obtained by the use of a more general diet if the sodium content could be reduced to the same level that is contained in the rice diet As a practical matter it is difficult to outline a general dietary regimen that will contain as little sodium as the Kempner diet When the strict rice and fruit diet is followed there is also a marked fall in the cholesterol in the blood Occasionally the chloride content also falls and with it a rise in blood nitrogen This then needs to be corrected by giving more salt

This diet is a very difficult one to take There are not many patients who will continue on it long enough to be of value Physicians who are enthusiastic about it and who impart their enthusiasm to their patients often succeed and claim very satisfactory results In my limited experience I have seen a few dramatic results, but in most cases the patients returned to what they regarded as a more palatable diet It is unlikely that the Kempner diet will eventually prove necessary In the meantime it has had useful results It reemphasized the importance of an extremely

low salt intake in the treatment of hypertension and congestive failure, a point that had been well known for many years It has also taught us that people can live comfortably for long periods of time on a very low protein intake, even if there is a nitrogen deficit, and it has focused our attention on the great importance of diet in relation to health and disease

CHRONIC MYOCARDITIS"

The term chronic myocarditis has formerly been used to denote a nonvalvular condition associated with heart failure There has been much discussion concerning this term and many substitutes offered Objection has been made to the term because myocarditis denotes an inflammation, and often no clinical or pathologic evidence of inflammation can be found At present, however as a result of the great advances in cardiac diagnosis many of the cases of so-called chronic myocarditis can be more accurately named and what was once a waste basket for a variety of conditions may now be reserved for only a rare occasion As one looks back at the cases so diagnosed in the past one will become aware of several clinical entities that now can be well recognized Some were instances of masked hyperthyroidism that were overlooked Others were instances of cor pulmonale or heart failure from chronic emphysema or pulmonary arterial disease Still others were cases in which the heart failure was due to avitaminosis (the so-called beriberi heart), or in which edema was present because of a low protein content of the blood A few were cases in which there was actual valvular disease, either mitral or more frequently aortic stenosis, in which the clinical signs were not elicited or were misinterpreted I know this has frequently happened in the past because subsequent examination would uncover a systolic thrill at the base of the heart or x ray examination later would disclose calcification of one of the valves, and finally because in some there was marked valvular disease on postmortem examination A large group of cases that formerly were called chronic myocarditis we now can recognize clinically as due to coronary artery sclerosis or coronary thrombosis The increased interest in angina pectoris and the great advances in electrocardiography have enabled us to make these anatomic diagnoses in many instances previously overlooked

There remains the large group that is now called hypertensive heart disease This does not mean that the mechanism of heart failure that develops in these cases is adequately explained on the basis of the hypertension The unknown factor X mentioned previously still has a bearing on the problem but we are enabled to classify such cases under the heading of hypertensive heart disease merely because there is or has been hypertension and there is heart failure without any other predictable anatomic abnormality When all these well recognized conditions and a few other unimportant ones like syphilis and anemia are carefully sought for and found absent, there still remains a very small group of patients in whom heart failure takes place There is no hyperthyroidism emphysema, valvular disease, coronary artery disease or hypertension and yet the heart muscle failed Such hearts on postmortem examination are generally en-

larged and may show an apparently normal myocardium The present methods of pathologic study fail to explain why the heart muscle was inadequate to maintain a normal circulation In a few there are found pathologic changes in the myocardium but no satisfactory cause for such changes These cases form the small remainder of what was formerly called 'chronic myocarditis"

Some of these cases which do show inflammatory changes in the myocardium, are called Fiedler s myocarditis, although the pathologic findings in this condition are not distinctive They generally are not very responsive to treatment and run a course lasting some months to a few years, rarely longer Occasionally the process may be essentially confined to the right ventricle and present a picture of right-sided heart failure

RARE FORMS OF HEART DISEASE

Myxedema Heart

With prolonged advanced myxedema certain changes take place in the circulation that are designated as "myxedema heart Such patients not only have the customary findings, such as a low metabolism, dry skin, coarse, scanty hair, a feeling of coldness, puffy appearance of the face, anemia and a high blood cholesterol, but show more than the usual edema of the legs and marked dilatation of the heart In most cases what is thought to be cardiac dilatation turns out to be pericardial effusion Rarely there may be fluid in the abdominal or pleural cavities In most cases there actually is no true congestive failure despite the cardiac dilatation Such patients complain of weakness and dyspnea but not of orthopnea The electrocardiograms show ventricular complexes of low amplitude (Figs 131, 180) The diagnosis is readily made, keeping the above features in mind

The important point regarding treatment is that patients with this condition do not respond to digitalis but can be cured by thyroid administration Thyroid medication should be given slowly and cautiously Too rapid increase in the metabolic rate can bring on acute left ventricular failure or angina pectoris A dose of ¼ to 1 grain of thyroid gland extract administered daily is often sufficient If anginal pain develops it may be best to accept only a partial recovery in myxedema, permitting the basal metabolic rate to remain about —10 per cent Under skillful management the heart returns to normal size, the electrocardiograms show normal tracings and all symptoms disappear

Heart Failure from Arteriovenous Fistula

There is one form of heart muscle failure which may properly be discussed in this connection, although it is in no way related to either infection or degeneration, i e, myocardial failure from a peripheral arteriovenous fistula Here the heart dilates and dyspnea and congestion may develop purely as a result of the increased work of the heart consequent to the short circuiting of the blood All evidence of cardiac disease may

promptly disappear after surgical removal of the fistula or aneurysm This condition is generally traumatic in origin and often follows a gunshot or stab wound of a limb The continuous murmur over the fistula or aneurysm, the pulsating veins distal to the lesion, the high pulse pressure and the prompt slowing of the heart rate that follows compression of the fistula are diagnostic of this condition It is one of the few types of heart failure from purely mechanical causes that is readily amenable to curative treatment

Not very long ago I learned of a case in which a nontraumatic arteriovenous fistula developed in the pelvis of a kidney The patient had had advanced heart failure for years and was bedridden Her physician, Dr F B Camp of Missouri, made the diagnosis by hearing a continuous murmur over the loin Even after being refractory to all ordinary medical treatment, the patient was cured by a nephrectomy

Ruptured Valves of the Heart

Occasionally one sees instances of rupture of the valves In most cases this occurs in previously diseased valves After a sudden strain a free aortic insufficiency may develop, especially in a preexisting syphilitic lesion, with a resulting loud or musical diastolic murmur Without any precipitating effort this complication may arise during the course of bacterial endocarditis Likewise, in old rheumatic disease of the mitral valve, ruptures may occur, but then they are more likely to take place in the chordae tendineae This apparently is more frequent than has generally been appreciated, even when no previous disease was present In such cases, either after some unusual effort or quite spontaneously, a loud apical systolic murmur of mitral insufficiency and dyspnea suddenly develop Cardiac symptoms may appear immediately or insidiously only after some years Males are more commonly affected than females All these patients eventually die of congestive failure although some have survived for months or many years The possibility of a ruptured valve must always be considered when a loud murmur and cardiac symptoms appear abruptly

Beriberi Heart

In the Orient it has long been known that heart failure could result from an inadequate diet The condition was called beriberi heart One form of this dietary deficiency appeared as peripheral neuritis and the other as congestive heart failure This condition is now known to be due to a deficiency of vitamin B_1 It may take the wet or dry form or be a combination of both Its occurrence in this country has been emphasized by the studies of Soma Weiss It is most frequent in chronic alcoholics and in others who have abstained from necessary foods for long periods of time There is reason to suspect that similar vitamin B deficiency with cardiac complications may be present in pregnancy and hyperthyroidism In the severe form the clinical picture is one of advanced heart failure, both of the right and left ventricular type The heart is rapid, generally

regular, but occasionally shows a transient auricular fibrillation and often has a gallop rhythm Dilatation may be considerable and yet complete restoration to a normal size results with recovery Systolic murmurs are frequent but diastolic murmurs are rare There may be a pistol shot in the peripheral pulse and an increased pulse pressure In fact, many of the features resemble those seen in thyrotoxicosis or arteriovenous fistula There often are considerable peripheral edema, engorged liver and pulmonary congestion Mural thrombosis of the cardiac chambers with resultant emboli may be present The electrocardiogram may show a diminution or slight inversion of the T waves in any of the leads, and lengthening of the Q-T interval The important point is *that* this condition does not respond to ordinary cardiac therapy or digitalis whereas recovery can be dramatic and complete following the administration of 10 to 20 mg of crystalline B_1 intramuscularly three times a day (*Fig* 179) The subsequent diet should contain meat, flour and yeast

Heart Failure with Acute Nephritis

Congestive heart failure with dilatation of the heart and the picture of either left or right ventricular failure occasionally occurs *during* acute nephritis These cases will show hypertension, pulmonary rales and peripheral edema, as well as evidence of active nephritis Electrocardiograms at this time may show changes in the ventricular complexes, especially in the T waves, that superficially resemble those seen with myocardial infarction A similar situation may develop during the toxemia of pregnancy The response to cardiac therapy is likely to be satisfactory and when recovery takes place the heart may be expected to be normal or essentially so Restriction of fluid and salt, digitalis and occasionally intramuscular injections of 1 to 2 gm of magnesium sulfate may be helpful

Cor Pulmonale

The right ventricle may fail as a result of increased pressure in the pulmonary circuit This may occur acutely as following a large pulmonary embolus (acute cor pulmonale), or more slowly as in chronic pulmonary emphysema, silicosis or pulmonary arterial disease (chronic cor pulmonale) These conditions have already been discussed (see Chaps 6 and 16) It must be emphasized, however, that great care is needed to distinguish the breathlessness that results from the pulmonary disease per se from that which follows heart failure The treatment and prognosis of the two states are quite *different*

Scleroderma Heart

It is now known that pathologic changes in scleroderma are not confined to the skin and subcutaneous tissues Cardiac complications occur consisting of disintegration of muscle fibers and extensive replacement by fibrous tissue This can result in cardiac enlargement and congestive failure

Auricular fibrillation, gallop rhythm and slight prolongation of the Q-T interval of the electrocardiogram have been noted in different cases I have seen instances in which because of related findings one case resembled chronic rheumatic heart disease and another thyrocardiac disease The treatment is the same as that for any ordinary case of heart failure The new Compound E and adrenocorticotropic hormone (ACTH) might well be helpful in this condition

Tumors of the Heart

Both malignant and benign tumors of the heart are very rare Metastatic tumors of the heart are about ten times as common as primary tumors The diagnosis of cardiac tumors is very difficult and most cases are first recognized at autopsy Peculiar x-ray silhouettes of the heart and the finding of bloody fluid in the pericardium not otherwise explained may lead one to arrive at a correct antemortem diagnosis Occasionally auricular flutter or fibrillation, transient or permanent, may be present with tumors of the heart, possibly as a result of involvement of the auricles The most common primary origin of cardiac metastases is carcinoma of the bronchus or lung There is one type of cardiac tumor that has particular interest This is a myxoma of the left auricle It is a benign tumor that may produce signs resembling those of mitral stenosis Such signs may change from time to time apparently owing to alterations of the position of the tumor which may be pedunculated The resulting events are similar to those seen in rare cases of ball valve thrombus in mitral stenosis It is not too much to hope that someone may successfully diagnose this type of tumor and attempt surgical removal

Cardiac Complications in Lupus Erythematosus Disseminatus (Libman Sacks Disease)

A group of conditions that are closely related if not different manifestations of the same underlying process, has come to light and has been described under a variety of terms Although there is still confusion in the terminology and complete lack of knowledge as to the etiology, the conditions we now recognize as lupus erythematosus disseminatus and non bacterial verrucous endocarditis (Libman Sacks disease) are known to involve the heart They are not infrequently associated with a sterile type of pericarditis and may show small nonbacterial vegetations on the valves of the heart In fact there are also rare instances in which the myocardium is involved as manifested by a prolongation of the P R interval in the electrocardiogram This group of diseases appears to be closely related in its pathologic findings to rheumatic fever, periarteritis nodosa and scleroderma, from which it may be differentiated at times, only with difficulty It also may be confused with subacute bacterial endocarditis because of many features that are common to both diseases

It must be appreciated that lupus occurs almost solely in females during the years of menstruation, may run a prolonged course of months

or years, may show no lesions on the face at the start or for long periods during its course and can display cycles of high fever with intervening periods of quiescence There is generally a leukopenia or at least an absence of leukocytosis and it may be associated with an increase in the globulin content of the blood The heart may be spared but often is involved, as are other serous linings, resulting in pleuritis, peritonitis, pericarditis or endocarditis There generally is albuminuria and often pulmonary consolidation The diagnosis is likely to be overlooked until the characteristic butterfly rash appears over the nose and cheeks The disease is almost invariably fatal

At present there is no known effective treatment, although spray x ray treatment is being tried for cases of lupus erythematosus disseminatus with what appears to be favorable temporary alleviation of symptoms Here also there is reason to believe that Compound E or adrenocorticotropic hormone (ACTH) will favorably affect this condition

The Heart in Addison's Disease

With the discovery of effective methods of treatment for Addison s disease by means of synthetic desoxycorticosterone, cortical extract and sodium chloride, a new type of heart failure is now met with The ordinary patient with Addison's disease has a small heart, often much smaller than normal Under this new treatment, the heart returns to a more normal size In some cases the heart is not able to make the necessary adjustment to the rapid changes in dynamics, and with the increase in blood volume and blood pressure, the retention of salt and undue dilation of the heart, pulmonary edema, peripheral edema and even rapid fatality may result Therefore, if any of the signs of heart failure appear in a patient under treatment for Addison's disease the medication should be omitted for a while and then reinstituted in smaller doses It should be appreciated that it is the synthetic desoxycorticosterone, which is the salt retaining factor, and the sodium chloride, rather than a preparation such as eschatin, which is the whole cortical extract, that causes the above cardiac complications

Familial Cardiomegaly

Recently William Evans of London described a small group of ca es that seem to form a clinical entity Such patients may have slight or no symptoms in their early years Later palpitation, giddiness and frank Adams-Stokes syncopal attacks or transient auricular fibrillation may occur Death may take place suddenly without congestive failure The heart will show very little except enlargement but the electrocardiograms may reveal various arrhythmias such as premature beats, paroxysmal tachycardia, auricular flutter, auricular fibrillation, auriculoventricular block or intraventricular block The condition is met with in children or young adults, and what is particularly characteristic is that more than one member of the same family are afflicted The cause is unknown and it is not thought to be due to glycogen deposits

Heart Failure from Deformity of the Chest

Marked deformity of the chest, especially following poliomyelitis or tuberculosis of the spine, may eventually lead to heart failure At first only slight dyspnea on exertion or with change of bodily position may be present The vital capacity of the lungs becomes considerably diminished, while the residual air increases in proportion As the condition progresses, less and less oxygenation of the blood takes place With this there may be attacks of palpitation, weakness, cyanosis, tendency to faintness or unconsciousness, and marked dyspnea Such patients quickly succumb to pulmonary infections The heart is not much enlarged, though there is often right ventricular hypertrophy The rhythm is generally regular and the pulmonary second sound is accentuated The velocity of blood flow, the venous pressure and the cardiac output per minute are within normal limits Once major symptoms develop the patient s activities become markedly restricted and the downhill progress does not seem to be influenced by cardiac treatment

Ordinary cardiac therapy is on the whole ineffective in this type of cardiopulmonary failure The only hope is preventive treatment These chest deformities produce harmful results if they occur during the growing years of life Every effort should be made to prevent or relieve these distortions of the chest when the child is young The orthopedic surgeon should be called in to do whatever he can in order to lessen the deformity When cardiac symptoms are already present, exercise should be greatly restricted and respiratory infections avoided as far as possible Oxygen and ephedrine may be helpful, and morphine should be avoided

THE HEART AND ACUTE INFECTIONS

It has become more and more evident that many acute infections are associated with some injury to the heart muscle In most infections whatever damage to the heart may take place must be inconsequential for very little clinical evidence becomes apparent during the acute infection and no residual cardiac defects persist afterwards However careful and frequent electrocardiographic studies during infections like diphtheria, typhoid fever, pneumonia, mumps, meningococcus meningitis mononucleosis and many other infections have shown changes in the ventricular complexes or the conduction mechanism These are generally transient, with complete restoration to normal Likewise in the occasional fatal case of some of these infections, pathologic evidence of inflammation of the ventricular musculature (acute myocarditis) has been detected Inflammatory lesions in the myocardium have been found in virus diseases such as scrub typhus poliomyelitis, encephalitis, infectious hepatitis and others It has long been known that with diphtheria, rheumatic fever and Chagas disease the heart muscle frequently shows extensive inflammatory injury, and heart block and electrocardiographic abnormalities are quite common It is now clear that although in most cases this type of acute myocarditis has little clinical or practical significance occasionally it may be the direct cause of death What is not known is whether some of the cases of

subclinical acute myocarditis which are even undetected in ordinary practice form the background for subsequent damage to the heart muscle In other words, can some cases of unexplained cardiac hypertrophy or so-called chronic myocarditis of unknown etiology be the end result of early injury sustained during an otherwise harmless acute infection? There is more than a suspicion that this is true I am increasingly convinced that diphtheria, which frequently is associated with heart block during the acute stage and is then generally fatal, may be the initial cause of complete heart block and Adams Stokes disease which occur many years later There are probably other instances in which an acute myocarditis during an infectious illness tells its tale many years later in some form of heart muscle disturbance

As an illustration of the frequency of mild involvement of the myocardium during acute infections one can cite the experience with mumps Ordinarily little attention is paid to the heart in this disease No one has associated significant cardiac complications with parotitis However, in a series of 100 cases 15 per cent showed definite abnormalities in the electrocardiograms, mainly T wave changes, and two showed complete heart block All the abnormalities disappeared with recovery from the infection The injury to the heart muscle was apparently mild and reversible

The following is an instance of acute myocarditis accompanying virus pneumonia A woman 34 years old who previously was well, never having had hypertension or rheumatic infections, developed ordinary influenza The virus was isolated in this case and found to be type A She quickly developed alarming cardiac symptoms She complained of substernal tightness, showed pulmonary edema and weak pulse There was a leukocytosis of 20,000 and the x ray of the chest was compatible with atypical pneumonia The electrocardiograms displayed bizarre complexes resembling potassium intoxication There were slurred broad QRS complexes and complete auriculoventricular dissociation with a ventricular rate of about 90 Within one week she died Postmortem examination showed extensive interstitial round cell infiltration and much degeneration of muscle fibers The coronary arteries and valves were normal The heart weighed 480 gm although she was known to have had a normal sized heart on x ray examination two months before

Cases similar to the above must be occurring either in a fatal form or with less extensive damage so that recovery takes place In the past the virus origin has not been identified and it is still a difficult laboratory procedure, so that absolute proof of the nature of the acute myocarditis will rarely be established It is reasonable to suppose that milder forms of acute myocarditis frequently go unnoticed in virus and other infections

Unfortunately therapy is likely to be of little value in this type of acute myocarditis Obviously the early and adequate use of specific measures when available, such as antitoxin for diphtheria, penicillin for pneumonia and aureomycin for virus infections, will either prevent or minimize the heart muscle injury It is doubtful whether digitalis preparations will be helpful The state of shock may be combated with transfusions of blood

8

Thyroid Heart Disease

Hyperthyroidism, whether due to a diffuse hyperplasia or to a toxic adenoma of the thyroid gland, produces certain disturbances in the circulation It has been the prevailing opinion that hyperthyroidism alone does not cause permanent structural changes in the heart and that when there is gross evidence of congestive heart failure or angina pectoris, other independent conditions will be found such as hypertension, valvular heart disease or coronary artery disease A recent review of a considerable number of thyrocardiac patients, however, revealed many instances of advanced heart failure in which no cause other than thyrotoxicosis could be found and in which complete recovery occurred after appropriate treatment Frequently arrhythmias occur that are very troublesome to the patient which are purely the result of the toxic thyroid gland, and it may be expected that these will disappear if the hyperthyroidism is cured There are many changes that result from hyperthyroidism, independently of other conditions, which are similar to those seen in ordinary forms of heart disease Inasmuch as thyroid and other forms of heart disease frequently occur together in the same individual and the evidence of an overactive thyroid gland may be very obscure, it becomes extremely important to be familiar with the methods of diagnosis that serve to differentiate the one from the other This subject is one of the most important aspects of all heart disease for it comprises the one large group of cases in which the difference between accurate and inaccurate diagnosis and treatment is the difference between chronic invalidism or death and restoration of health and life

SYMPTOMS

We are all familiar with the typical picture of exophthalmic goiter It is not the purpose here to discuss in detail but rather to mention these features briefly The main emphasis is to be directed at the group of cases called "masked thyrocardiacs At the outset it must be appreciated, just as is true in many diseases, that no single sign or symptom is invariably present but that *even in the obscure cases the composite picture is sufficiently* suggestive to enable one to suspect the proper diagnosis The cardinal diagnostic points of hyperthyroidism are exophthalmos, thyroid

enlargement nervousness and palpitation When exophthalmos and a palpable thyroid are absent, as is the case in this particular group of patients, the other symptoms and signs of an overactive gland become more important because only by constant search into these features will the correct diagnosis be made It must also be borne in mind that these patients, who are nevertheless suffering from hyperthyroidism, come to the phys ician complaining of the same symptoms as do other patients with heart disease but without hyperthyroidism Their primary complaints will be shortness of breath, palpitation weakness, chest pain coughing swelling of the legs or abdomen Inasmuch as they often lack the obvious evidence of thyroid disease and have evident organic heart disease of one form or another they are treated for the latter with little if any success and the former is entirely overlooked

It is always necessary to have the possibility of masked hyperthyroidism constantly in mind when treating cardiac cases This is especially true if certain peculiar clues are detected Of first importance is transient auricular fibrillation Although this phenomenon occurs in other conditions, of none is it so characteristic and in none does it occur so commonly as in hyperthyroidism It, therefore, should be the invariable practice to suspect a toxic thyroid gland whenever this arrhythmia is observed even when other forms of heart disease such as mitral stenosis are also present Undue loss of weight, nervousness tremor of the fingers and excessive perspiration are frequently not investigated with sufficient care when present in cardiac patients They are too often attributed to the custom ary forms of heart disease with which they are frequently associated, whereas in some cases a latent hyperthyroidism proves to be the more important cause This is particularly so if the loss of weight takes place in the presence of a fair or good appetite Other common findings in these patients are transient glycosuria and transient periods of mild diarrhea or hyperactivity of the bowels These patients, therefore are at times treated for diabetes or for some gastrointestinal disorder The diabetes here rarely is of much importance the patients often stating that they have had to pay very little attention to their diet and that it did not *seem to matter very much whether they followed a diet or not It is also* of some interest that whereas the ordinary inactive cardiac patients gen erally need cathartics those suffering from latent hyperthyroidism as a rule do not

The general appearance of the patient often has given the first suspicious evidence that a latent hyperthyroidism long overlooked, was present The skin takes on a peculiar appearance, rather difficult to describe, that I have called a salmon colored hue It is warm, moist, hyperemic and slightly pigmented The eyes, in the absence of gross exophthalmos, may show a peculiar stare Sometimes this stare is unilateral There is also a certain quickness of motion, an alertness and even an attractiveness to the behavior of many of these patients that one is not accustomed to find in the ordinary case with heart disease In fact most patients suffering from

heart failure, particularly when more or less bedridden, are sluggish both in mind and body, whereas with the same degree of decompensation those who also have hyperthyroidism often retain this peculiar alertness and comeliness It is also common to learn that these patients prefer cold to warm weather In addition many have premature gray hair Often this has preceded by many years the development of the major circulatory symptoms Incidentally the appearance of premature, rather striking, grayness of the hair has often been an important clue in the diagnosis of other conditions Early graying occurs in four main groups of individuals First there is a group of perfectly normal people who begin to have gray or even white hair in the twenties or thirties Often they have a family history of a similar tendency Among these will be a considerable number who al o have a tendency to premature arteriosclerosis Then there are three diseases with which it is not infrequently associated, i e, asthma, hyperthyroidism and pernicious anemia Although the exact causal relationship is obscure it may turn out that early graying of the hair is due to one common abnormality of the endocrine system Needless to say, such features will not be ascertained or appreciated unless the physician has *already been suspecting the thyroid gland It is very striking in hospital* records how the history and physical findings change after the routine admission notes have been made, when the same observer is told that the patient may have masked hyperthyroidism Only then do the observations appear that the skin is moist and somewhat pigmented and that the patient has preferred cold weather or that there has been a hyperactivity of the bowels In other words, one has to seek the evidence, for the patient may only be interested in his breathlessness

It is to be expected that patients with thyrotoxicosis would be prone to avitaminosis Even with a normal dietary intake, which is not adequate because loss of weight occurs, increased metabolism demands still greater amounts of vitamins The result is that deficiencies are common This may manifest itself in the form of a red sore tongue, skin changes or red palms Possibly some of the cardiac abnormalities in hyperthyroidism are actually the result of vitamin B_1 deficiency

The examination of the circulation and especially the heart is of the greatest importance in these cases for not only are there peculiarities to be found that help in arriving at the correct diagnosis, but here are al o the abnormalities that are associated with the common forms of heart disease with which hyperthyroidism is confused It is necessary to repeat that transient auricular fibrillation is very common, but many patients even develop the permanent form of this irregularity The character of the heart sounds is very apt to be hyperactive An accentuated first heart sound is common in mitral stenosis, in anemia, in some cases of hypertension, in cases of nervous or unstable hearts but especially so in hyperthyroidism With this hyperdynamic contraction, often detectable on fluoroscopic examination, there is a diffuse and snapping apex impulse The vibration of the chest wall resulting from this hyperactive heart

makes the impulse diffuse and often creates the impression that the heart is larger than is actually the case Furthermore a vibration is set up in the chest wall which on palpation feels like a thrill

When in addition one appreciates the fact that in many cases of hyperthyroidism there is present a systolic murmur either at the apex or base of the heart, one can readily see how easily this condition may be confused with mitral stenosis This mistake has often been made by most competent physicians In hyperthyroidism the x ray may even show a slight prominence in the region of the left auricle similar to that which occurs in early mitral stenosis, although the prominence is both the result of dilatation of the pulmonary vessels and the left auricle In both conditions symptoms of cardiac disease exist auricular fibrillation is common, both have an accentuated first heart sound and a systolic murmur and in both there may be a palpable thrill To be sure the thrill occurs during diastole in mitral stenosis and with systole in hyperthyroidism, but when the heart rate is rapid it is often impossible to time the palpable thrill with any accuracy The preconception that the condition ought to be mitral stenosis often leads the observer into the false timing of the thrill as presystolic The one difference between the two conditions is that no murmur will be heard during diastole in cases of hyperthyroidism This differential point is of the greatest importance The difficulty becomes greater still when both conditions exist in the same patient as not infrequently occurs, for one then finds ample evidence of mitral stenosis and has to rely on other criteria to make the additional diagnosis of hyperthyroidism

There are further peculiarities of the circulation in hyperthyroidism that require mention It is often difficult and may be impossible to slow the heart rate by the use of digitalis when auricular fibrillation is present Ordinarily with auricular fibrillation, especially when there is no fever, one expects a specific slowing to an essentially normal rate when adequate doses of digitalis are administered In fact, when such slowing does not occur one is justified in suspecting that the drug is not of normal potency or that the patient has hyperthyroidism The failure to obtain the expected slowing of the apex rate of the heart in cases of auricular fibrillation following appropriate digitalis dosage has been, in several instances the first *clue that a masked hyperthyroidism was present Slowing is promptly* obtained if the basal metabolic rate is reduced or brought to normal by the use of iodide medication or surgery

In some puzzling cases the determination of the velocity of blood flow helps to decide whether the cardiac symptoms are due to thyrotoxicosis or not In most cases of ordinary congestive failure the circulation time is slow, i e, over twenty seconds In hyperthyroidism the velocity is apt to be fast, even in the presence of congestive failure The only other conditions in which the velocity of blood flow is rapid are beriberi heart, auriculoventricular fistula, anemia and fever If, therefore a cardiac patient in failure has a reading of twelve to fifteen seconds it is strong evidence in support of the diagnosis of thyrotoxicosis One other laboratory test

may be helpful, i e, the cholesterol content of the blood This is frequently decreased to about 130 to 150 mg per 100 cc whereas most ordinary cardiac patients will show readings of over 200 mg

Blood Pressure

Another aspect of the circulation in hyperthyroidism is the blood pressure The systolic reading may be normal or slightly elevated In the cases that are occupying our particular attention at present, the so-called ' masked thyrocardiacs, the systolic pressure is frequently elevated This is due to an independent vascular hypertension for it remains essentially unchanged when the hyperthyroid state is eliminated What is more characteristic is an increase in the pulse pressure, not so striking but similar to that observed in aortic insufficiency Readings of 160 mm systolic and 75 mm diastolic are not uncommon There may be other features resembling aortic insufficiency such as a capillary pulse, Corrigan pulse, pistol shot in the femoral artery and even the Duroziez's sign These are all the accompaniments, if not the result of, the high pulse pressure and the peripheral vascular dilatation Finally the heart rate itself is generally rapid At times it is difficult to distinguish the tachycardia of hyperthyroidism from that of neurocirculatory asthenia One helpful distinction lies in the fact that during sleep the rate in the latter condition is normal while in the former the tachycardia, although less pronounced, will still persist It must not be expected that a rapid heart will be found in all cases of hyperthyroidism The rate may be under 80 and even under 70, especially in males, in the presence of a toxic gland I have repeatedly seen such cases where because of the slow heart rate hyperthyroidism had previously been entirely overlooked

Basal Metabolism

When the possibility of hyperthyroidism has arisen the basal metabolic rate should be determined Unless the patient is in great distress this can generally be accomplished satisfactorily The greater difficulty is to interpret the readings If it is found to be distinctly below normal an active hyperthyroidism can be ruled out If the figures range from zero to +10 or +15 per cent it is extremely unlikely that the gland is toxic There are rare instances, however, in which an active hyperthyroidism is going on, in the sense that cardiac disturbances take place as a direct result of the thyroid gland, while the basal metabolic rate is perfectly normal I know of two patients in whom transient attacks of auricular fibrillation were taking place who had a perfectly normal basal metabolic rate During the course of the subsequent several months, while the attacks of palpitation were recurring, the rate gradually rose to +20 per cent and later to over +40 per cent Then, after subtotal thyroidectomy, the metabolic rate fell to normal and the paroxysmal fibrillation disappeared It is evident in such rare cases that the thyroid was toxic, as far as its effect on the heart was concerned, at a time when the metabolic rate was normal May it be that patients such as these actually had an original

metabolic rate of −20 per cent ≠ (which many healthy individuals have) and when an elevation of 20 per cent occurred they were already ' toxic and yet showed normal readings?

When the readings are +20 per cent to +30 per cent, it may be no simple matter to decide whether this slight elevation in rate is due to the thyroid gland or is the result of the cardiac failure itself Slight to moderate elevation in the metabolic rate has occasionally been found by some observers in cases of cardiac failure These borderline readings may be very difficult to interpret When properly conducted and repeated metabolism determinations show an elevation of +35 or more, in the absence of fever or leukemia, one can be fairly certain that they indicate hyperthyroidism There is a small group of cases, especially associated with hypertension, in which even this degree of elevation in the basal metabolism will be associated with a normal thyroid gland An elevation of the metabolism in a hypertensive patient should also make one think of the possibility of an adrenal tumor (pheochromocytoma), especially if the blood pressure falls and the pulse rate rises on standing In the ordinary hypertensive patient the pressure rises somewhat on standing I have also seen four patients with aortic stenosis who had readings persistently over +20 to +40 per cent and yet had normal thyroid glands when examined grossly and microscopically Tests need to be repeated since not infrequently the initial elevation disappears without specific medication when a second or third test is made The specific reduction of the basal metabolic rate following iodine administration can serve as a valuable aid in diagnosis Properly conducted and properly interpreted this laboratory test proves to be of indispen able value in the diagnosis of many of these obscure thyrocardiac patients Another test that would indicate hyperthyroidism is the finding of an elevation of protein bound iodine in the blood

On very rare occasions hyperthyroidism may be associated with Addison s disease The diagnosis is then difficult It may be suspected on the basis of the following features The absence of hypoglycemic attacks, which are frequent in Addison s disease, would make one think of thyrotoxicosis The heart will not be small and the basal metabolism will be relatively elevated, i e , more than −5 per cent Furthermore, the clinical manifestations of hyperthyroidism will be atypical and not prominent

The most recent tset for thyrotoxicosis is the radioactive iodine uptake determination of the thyroid gland This is performed by the use of a Geiger counter which determines the amount of iodine that accumulates in the thyroid gland during a few hours after the administration of a given amount (0 1 millicurie) of radioactive iodine Cases of myxedema will show strikingly low figures and those of hyperthyroidism will show high uptakes This test is very helpful because it requires very little cooperation on the part of the patient and the result is obtained directly The test is invalid and may lead to a false diagnosis if the patient has had iodine in his food or as a medication before it was done In fact, I recently saw a patient in whom the diagnosis of thyrotoxicosis was made

clinically which was confirmed by operation, and yet the iodine uptake test was normal If inorganic iodine such as potassium iodine or an organic compound like lipoiodine has been taken some weeks, some months should elapse *before the test may be regarded as valid*

TREATMENT

Until several years ago the choice of treatment for this group of patients was a fairly simple matter It consisted of all the usual procedures employed in the treatment of the common forms of heart disease, but in addition giving iodine After a period of ten to fourteen days on this management a subtotal thyroidectomy would be performed and the patient would do well The details of this program will be discussed below The situation is now changed because there are two new nonsurgical methods that are available, i e, thiouracil preparations and radioactive iodine There is abundant experience with the earlier type of treatment, extending over a period of many years It will take some time before a comparable experience will be accumulated in order to determine the relative efficacy of these newer methods of treatment

Radioactive iodine has already been successfully employed in thyrotoxic states The procedure is simple and inexpensive, would require very little hospitalization and carries no immediate risk, but at present it is not available except to a limited number of centers The principle involved is clear The iodine is taken up by the thyroid gland and only to a very little extent by any other organ There the radioactivity takes place in intimate relation to the thyroid cells to produce their destruction or decreased activity The result is similar to the effects of x rays, except that no other organs are appreciably involved It is too soon to appraise the real value of this treatment, but already cures have been obtained, and *in fact myxedema has been produced when the dose was excessive* How permanent the cures will be and what late complications in the thyroid gland or other organs may ensue remains to be seen We wait hopefully for further development of this method

The other nonsurgical method is the use of propylthiouracil Since the introduction of thiouracil by Astwood as a method of decreasing thyroid function newer less toxic preparations have become available These drugs block the formation of thyroid hormone within the gland The one most commonly employed at present is propylthiouracil With this drug serious complications such as agranulocytosis are very rare Furthermore, there has been considerable experience with its use, though the time covered is still far short of that available from the older method, i e, iodine and operation

It has also become clear that the underlying cause of hyperthyroidism is not likely within the thyroid gland itself The thyrotropic hormone of the pituitary gland has much to do with the thyrotoxic state It is also thought that exophthalmos is determined by some control of the pituitary gland *Certain patients may be cured of hyperthyroidism and yet progress* with malignant exophthalmos In these cases the administration of thyroid

gland extract and iodine may be helpful Very recently adrenocorticotropic hormone (ACTH) has also been used with some success It is therefore not surprising that attempts at therapy should develop which involve a direct attack at the production of hormones by nonsurgical measures

After the diagnosis has been established propylthiouracil is given orally The early doses used to be 50 mg three times daily, but more recently this has been increased in most cases to 100 mg three times daily The condition is followed particularly by repeated basal metabolism determinations Generally it requires several weeks and sometimes much longer to lower the metabolism to normal Occasionally the drug is ineffective or only partially so Medication needs to be continued for some months If a satisfactory result is obtained the question will then arise whether to omit the drug entirely or to decrease the dose This can only be determined by trial and error In some cases, after a period of months the condition appears to be arrested even after complete withdrawal of the medication In others symptoms recur

As years have elapsed some of the early enthusiasm has waned as more and more of these cases have required ultimate operations As I have watched this development it has appeared that many patients under propylthiouracil treatment have required more lengthy hospitalization and observation than with the older method of treatment There can be no doubt however, that a large number can be successfully treated in this way, if careful observation is carried out I have had patients with auricular fibrillation and congestive failure regain compensation with restoration of a normal sinus rhythm Likewise I have witnessed dramatic relief of angina from the use of propylthiouracil alone It is striking that in several of such cases the anginal attacks, which were recurring as often as ten or more times daily at rest, disappeared within a few days after the onset of drug therapy This striking change took place therefore before there was any appreciable fall in the basal metabolism This makes one think that the effect was produced by a neutralizing action on some toxic product rather than by decreasing the work of the heart In general it is clear that propylthiouracil or similar compounds have great value in a selected group of patients suffering from thyrotoxicosis and even in thyrocardiac patients, and will spare some patients the need of surgery

I now come to the discussion of the older method of treatment This consists of giving 10 drops of Lugol solution three times daily in addition to the measures ordinarily employed in the particular cardiac case Digitalis and diuretics would be used if there is auricular fibrillation or congestive failure as well as thoracentesis and other procedures as necessary In most cases a period of seven to ten days will be required to prepare the patient for a subtotal thyroidectomy Occasionally because of the severity of the complicating heart disease, a longer period will be necessary The diet should be abundant and it is well to add vitamins, especially vitamin B_1 The preoperative medical treatment should be continued as long as there is evidence of a progressive improvement as shown by a fall in

the metabolism, slowing of the heart rate, and a diminution in congestion It is useless to wait for the auricular fibrillation, if present, to disappear or to use quinidine preoperatively The type of anesthetic does not seem to be of any great importance Many of my patients were given a general anesthetic although others had local anesthesia The surgeon should be urged to take out more rather than less of the gland, as too frequently an insufficient amount is removed

The postoperative care consists of continuing iodine therapy for about two weeks, and whatever other measures the cardiac problem requires If the rhythm of the heart had been regular before the operation an attack of transient auricular fibrillation the first day or two after the operation is not uncommon If there had been persistent auricular fibrillation, in many instances the heart will be found to resume its normal rhythm spontaneously during the first week or two postoperative The basal metabolic rate should be determined from time to time with the hope that it will be normal within two weeks after the operation In most cases in which satisfactory results are obtained the metabolic rate is already normal by that time, although in some it is still appreciably elevated only to fall to normal more gradually several weeks later If the rate remains elevated the operation has not been a success and some of the troublesome features such as auricular fibrillation may persist If the metabolism has returned to normal and this irregularity is still present one should seek for other causes of auricular fibrillation, especially mitral stenosis Sometimes the murmur of mitral stenosis or aortic insufficiency may become audible only after the metabolism has been brought to normal after the operation On the other hand, a systolic murmur so frequent during active hyperthyroidism, often disappears with a return of the metabolism to normal On several occasions I have found definite evidence *of mitral stenosis which was previously unsuspected in patients who had* been operated upon for hyperthyroidism, where auricular fibrillation persisted despite a normal metabolism If this irregularity has not disappeared by a fortnight after the operation one may give quinidine with the expectation that a normal rhythm will be reestablished It is advisable to do this, bearing in mind that if mitral stenosis is also present, quinidine therapy will entail the same limitations and same dangers that obtain in cardiac patients without thyroid disease

The results obtained by the above method of treatment are nothing short of miraculous Patients previously invalided and apparently hopelessly so have been restored to good health and frequently to a state of complete recovery When it is appreciated that in many of these patients, those included by the term "masked thyrocardiacs, proper diagnosis could not even have been made up a few decades ago and that they would finally have succumbed to heart failure, one can then fully sense the progress that has been made in recent years This progress has been the combined result of the pioneer physiologic work in bodily metabolism, the wonderful development of surgical technic, and finally the careful bedside

clinical observations that brought to light this hitherto unrecognized group of thyrocardiac patients

The surgical mortality in these cases since the introduction of the preoperative use of iodine has become almost nil There is practically no cardiac patient too sick to be helped or too sick to undergo the operation Npt only is the immediate risk very slight but the improvement obtained is quite lasting Unlike other forms of chronic heart failure, where the improvement after careful treatment is apt to be only too temporary, here permanent relief is obtained and the patients often are able to carry on full duty without further medication To be sure, they are left with whatever independent form of heart disease, be it valvular, hypertensive or coronary, which happened to be present, but now the previously embarrassed heart resumes its compensated state and is able to continue so for years In no other group of patients who suffer from chronic intractable heart failure despite good medical management have the members been able to resume varying degrees of physical activities for as long a time as those in this group under discussion It is this difference in outlook and in treatment that makes the problem of thyrocardiac disease so important

One of the advantages of surgery is the detection and extirpation of any neoplastic process that might be present in the gland Occasionally one finds such cancerous tissue unexpectedly One never needs to worry about cancer of the thyroid in the presence of diffuse hyperplasia or classical Graves disease Malignant lesions occur exclusively in an adenomatous goiter, most commonly in a single nontoxic adenoma and only very rarely with the toxic type

There is no better way of crystallizing the above problem than to cite briefly some personal experiences One of the first patients of this type that I had the good fortune to examine in 1921 was a woman in the sixties who had had progressive hypertensive heart failure for two and one-half years, during the last six months of which she had been bedridden Despite excellent care under the supervision of competent consultants she had been progressively losing ground The clues in this case were a history of transient auricular fibrillation progressing into the permanent form of the irregularity a recurrent glycosuria and the failure of the rapid irregular heart to slow on adequate digitalis dosage In addition there was a certain quickness of motion that the patient manifested despite the fact that she was orthopneic, showed free fluid in the chest and abdomen and had generalized anasarca The basal metabolic rate was found to be +71 per cent and eventually, after a subtotal thyroidectomy all signs of congestion disappeared, the rhythm of the heart became regular spontaneously, the metabolic rate became normal and during a subsequent period of twelve years she never had return of the heart failure After a long and useful life she finally developed dyspnea and hypertensive heart failure and died about fourteen years after the operation Her condition remained long overlooked because of the absence of thyroid enlargement and exophthalmos

Another dramatic result was obtained in the case of a man 60 years old suffering from angina pectoris During a period of six years there had been increasing occurrence of the attacks so that finally they came very frequently while the patient was at rest, even awaking him from sleep every half hour or so The condition remained refractory to all medical methods of treatment although the attacks were always temporarily relieved by nitroglycerin In desperation the patient wanted to undergo some form of surgical operation such as cervical sympathectomy or alcohol injection of the dorsal roots to obtain relief He was in this state when I first saw him The physical examination revealed no abnormalities The heart rate ranged between 70 and 80 After a period of observation of a week during which time I was at a loss as to what to do, for he was having forty attacks of angina a day, it suddenly struck me that his skin was somewhat moist and slightly pigmented He also moved rather quickly and jerkily in bed This led to the suspicion of masked hyperthyroidism The basal metabolic rate was found to be +45 per cent and on Lugol s solution it fell to +5 per cent With this fall came a striking improvement with no attacks at night and only about four during each day No evidence *of an enlarged thyroid gland could be made out by palpation or x ray* examination Despite this a very small adenoma was found and removed from behind the manubrium This emphasizes the importance of x ray examination in searching for a mediastinal goiter when none can be felt in the neck Often one can be discovered in this way which would otherwise be overlooked All attacks of angina disappeared after operation and he was able to resume his normal activities for many years thereafter He experienced anginal attacks only if he hurried up hill In other words, the anginal state was actually present but was held in abeyance as the metabolism was kept normal

The following experience illustrates the difficulty of recognizing hyperthyroidism in the presence of mitral stenosis This man 40 years old, had a history of rheumatic fever and was known to have mitral stenosis He gradually developed increasing dyspnea and over a period of eighteen months became bedridden with advanced congestive heart failure His physician and the consultant in attendance had observed that it was impossible to slow the heart rate below 100 even when full doses of digitalis were given When I saw him it was this rapid irregular heart rate in the face of constant administration of digitalis together with a salmon-colored skin that first made me suspect a latent hyperthyroidism While in the state of advanced congestive heart failure with an apex rate of 170 to 180 the basal metabolic rate was found to be +40 per cent On the same dose of digitalis (0 1 gm twice daily) but with the addition of 10 drops of Lugol s solution three times a day, the heart rate quickly fell from 180 to about 60, the basal metabolic rate to +6 per cent and there took place an extraordinary diuresis Within ten days the patient felt perfectly well It was believed, however, that the improvement would only be temporary if the toxic gland were not removed There was no exophthalmos or palpable thyroid gland It was difficult in this case, as

in many others, to convince the patient that he should undergo an operation In fact it was not simple in some instances to prevail upon the surgeons to perform the operation, for often they did not believe the gland was diseased However after the operation in this case the striking improvement that had taken place on Lugol s solution was maintained despite the fact that the patient still had mitral stenosis and auricular fibrillation He was able to resume his work which he had been forced to give up two years previously

In another case a middle aged woman was seen in advanced congestive failure She had been quite sick and bedridden for several months and had had repeated tappings of the right chest, each time removing a liter or more of fluid The findings strongly pointed to the diagnosis of mitral stenosis The heart was quite rapid, grossly irregular, and the sounds were hyperactive There was a slight apical systolic murmur and a questionable faint diastolic murmur In addition there was a short vibration or thrill at the apex, difficult to time because of the rapid rate The electrocardiogram showed definite right axis deviation and the left auricle was found to be moderately enlarged on x ray examination One can see that all the features of mitral stenosis were present except for a clear-cut diastolic murmur After a week had elapsed our studies revealed that the blood cholesterol was low (140 mg) and the circulation time was fast (12 sec) despite the presence of marked right hydrothorax and enlarged liver The condition improved somewhat on iodine therapy and the metabolism fell *from +47 per cent to +12 per cent After a subtotal thyroidectomy further* improvement occurred, but auricular fibrillation continued Two weeks after the operation quinidine restored the rhythm to normal and the patient recovered completely Even the dilated left auricle and the right axis deviation returned to normal She has remained well and shown no signs of heart or thyroid disease these past nine years

One could go on at great length and describe numerous other instances in which apparently hopeless cardiac invalids were resurrected by, first *the detection of suspicious evidence of masked hyperthyroidism and then* by the institution of proper treatment The important lesson from all this is constantly to suspect an undetected toxic thyroid gland when treating patients with cardiac disease Although a very few such patients will be sufficiently improved by the use of iodine therapy alone, the vast majority will do better to undergo a subtotal thyroidectomy

The question of x ray treatment for hyperthyroidism deserves a word of comment I have had little experience with it because it is time-con*suming and too often ineffective The fact that* operation is so safe nowa days makes surgery the method of choice In a series of ninety-nine patients with thyrocardiac disease many of whom were critically sick, even still showing congestive failure at the time of operation there was no surgical mortality whatever These figures speak for themselves

The reader may appear confused as a result of the above discussion of the treatment for thyrocardiac disease The truth is that the author is also somewhat in the same position Now that we have several possible methods

of therapy the choice is difficult Obviously, if a physician works in a locality where there is an appreciable surgical mortality from thyroid surgery or where aphonia, hoarseness or tetany are not infrequent complications or if incomplete operations are performed and recurrent hyperthyroidism is common, it would be preferable to advise nonsurgical procedures However, in these thyrocardiac patients, most of whom are elderly, it has appeared to me that postoperative thyroid storms are almost unheard of and that the fear of progressive exophthalmos is extremely slight Furthermore, the cosmetic aspect of surgery is unimportant in this group The choice will also be influenced at times by economic considerations Hospital cases under propylthiouracil treatment have required more lengthy periods of observations and were away from their work for a longer time than those subjected to operation Finally, the therapeutic use of radioactive iodine is being explored Already many successes have been achieved Only future experience will clarify the ultimate place this new method will occupy in our choice of treatment It must be said that good results are not restricted to one method of treatment In the course of the coming years, with increased experience, the criteria for the selection of one course of treatment or another will become established

9

Syphilitic Heart Disease

Syphilis affects the circulation in one of several ways There is a very rare condition in which it involves the peripheral blood vessels, producing a picture of malignant arteriosclerosis and hypertension The more important lesions are aortitis and aneurysm, aortic insufficiency involvement of the coronary arteries and of the myocardium It is much more common in males than in females and in the colored race than in the white race

Aortitis and Aneurysm

Although the spirochetes no doubt localize in the aorta a short time after the initial infection clinical evidence of syphilitic disease of the aorta ordinarily does not become apparent for many years (fifteen to twenty on the average) after the primary sore However, there are occasional instances in which this does occur within one to two years The favorite site is the first portion of the ascending aorta although any part may become affected For many years therefore, a syphilitic process may be going on in the aorta without any impairment in health or any abnormalities on physical examination When syphilitic aortitis first becomes detectable it will do so by producing a slight dilatation of the ascending aorta It will be difficult to elicit this by percussion X ray examination will be necessary and even then it is no simple matter to differentiate a luetic dilatation from one that accompanies hypertension or one that is due to arteriosclerosis

For the most part syphilitic aortitis of itself produces no symptoms When it does so there is pain in the chest that resembles somewhat that seen in disease of the coronary arteries The resemblance however, is only superficial for the pain is not particularly brought on by walking and is not so characteristically constricting in type as it is in angina pectoris Here the pain is more boring and more steady It is frequently more troublesome at rest or at night in bed or when the patient lies in certain positions Physical examination for the most part reveals no essential abnormalities A localized systolic pulsation may be seen or felt at the base of the heart, particularly in the second right interspace On auscultation there need be no murmurs or irregularities whatever or only a slight systolic murmur is present The aortic second sound often takes on a peculiar accentuated or metallic quality When there are no other more

definite evidences of syphilis it is extremely difficult to make a clinical diagnosis As has just been mentioned the x ray may be very helpful but often still leaves us in doubt It is now thought that the finding of calcification of the wall of the ascending aorta on x ray examination is strongly suggestive of a syphilitic process This sign has proved to be correct in a few cases recently observed even when the serologic test of the blood was negative Similar calcification of the arch or descending portion of the aorta, however, does not have this same significance The Wassermann test will be found positive in only about 75 to 85 per cent of the cases so that when negative it cannot satisfactorily rule out the diagnosis and when positive it may not reflect a syphilitic process in the aorta

When the syphilitic process in the aorta produces a true aneurysm the diagnosis is more readily established This takes the form of diffuse or sacculated dilatation When the latter is present one can safely assume that syphilis is the cause Aneurysm of the aorta produces symptoms mainly by pressure on neighboring structures and this will in a large *measure depend on the portion involved and the direction that its enlarge-*ment takes It may press on the trachea or either bronchus and produce cough (often with a brassy quality), a tracheal tug, or atelectasis of the lung It may expand backward and cause erosion of bone in the vertebrae and ribs This then becomes quite painful On the other hand, it may erode bone anteriorly through the manubrium and clavicles and present itself as a pulsating tumor in the front of the chest Inequality of the pupils and pulses or aphonia may result from pressure on nerves or large arteries Aneurysms may attain a considerable size and produce no noticeable symptoms merely because their growth does not happen to cause pressure on any of these important structures

Apart from the findings just mentioned an aneurysm often produces local pulsations in unusual sites Such pulsations are to be sought for by inspection and palpation over the base of the heart and in the upper back between the scapulae All the points referred to should be gone into when an adult complains of a boring pain in the chest and careful x-ray examination should be made It has been said that there are two types of aneurysm of the aorta—those with signs and those with symptoms One should add a third, i e, those with neither signs nor symptoms I have seen patients with syphilitic aortic aneurysms the size of an orange when there were no symptoms and no abnormal physical findings (except in the x-ray) *even after the exact location of the aneurysm was known*

The efficiency of the general circulation is not materially affected by syphilitic aortitis with or without aneurysm If the valves, the coronary arteries and the myocardium are spared there will be no evidence of congestive heart failure The largest syphilitic aneurysm I ever saw occurred in a patient who had a perfectly normal heart There never was evidence of cardiac failure and on postmortem examination the weight of the heart was less than 300 gm and the valves and myocardium were normal in every way The aneurysm was almost as large as a football and originated from a hole in the aorta 1 5 cm in diameter When congestive

heart failure is present with luetic involvement of the aorta some added factor will be found which acts deleteriously on the heart

The syphilitic process may extend downward and involve the mouths of the coronary arteries which open just at the root of the aorta This is not a rare complication and when it does occur all the possibilities exist that attend an inadequate coronary circulation Anginal attacks may occur and sudden unexpected death result Syphilis, therefore, produces angina pectoris by narrowing the orifices of coronary vessels It hardly ever involves the main course of the arteries as occurs in ordinary cases of angina There have been a few cases reported in which the mouths of both the right and left coronary arteries were completely occluded by a gradual syphilitic process The circulation through the heart had apparently been adequately maintained although no flow through the coronary arteries was possible The work of Wearn has emphasized the possible role that may be played by the thebesian vessels in maintaining the health of the heart Compensatory circulation through these small channels that open directly into the ventricular cavities probably explains how such patients could have carried on in life and may also account for the compensatory mechanism that goes on in nonsyphilitic cases of coronary artery disease

Aortic Insufficiency

Of greatest importance is the effect of the syphilitic process on the aortic valves, as this accounts for most of the disability and mortality in syphilitic heart disease The disease often extends downward and destroys the integrity of the aortic orifice It then produces aortic regurgitation just as occurs in rheumatic fever but, unlike the latter, it never results in stenosis of the aortic valve The appearance of the aortic cusps also differs in the two conditions In the syphilitic form the commissures become separated while in the rheumatic they become fused When aortic insufficiency is present the heart may become enlarged and in time congestive failure results Cardiac enlargement does not occur because of the aortitis but as a direct result of the valvular insufficiency When dyspnea and other evidence of circulatory failure intervene the prognosis is apt to be quite grave as response to treatment is on the whole unsatisfactory

The diagnosis of syphilitic aortic insufficiency will depend on eliciting evidence of this type of valvular defect (Chap 4) It is most important to appreciate that during the early stages, the aortic diastolic murmur which is so important may be very faint and only audible with the patient upright and after a forced expiration At times it is difficult to determine whether aortic insufficiency is part of a syphilitic, rheumatic hypertensive or arteriosclerotic process and will require all the means available to help in the differentiation The difficulty may be particularly great because an Austin Flint murmur resembling mitral stenosis rarely is present in syphilitic aortic insufficiency In general luetic aortic incompetency occurs at about the age of 40 to 60, is much more common in males than females, is only rarely associated with auricular fibrillation and never results in

stenosis of valves It is compatible with good general health and vigor for many years but when cardiac decompensation first develops the downward progress from then on is apt to be rapid Cheyne-Stokes breathing and nocturnal dyspnea are common

Syphilitic Myocarditis

Direct syphilitic involvement of the heart muscle is very rare When it does occur it may take one of two forms There are isolated instances of localized gumma of the heart When such a process involves the conduction apparatus, complete heart block may result There are rare instances in which a myocardial gumma becomes calcified or causes a localized aneurysm Under such circumstances, x ray and kymographic examination may help in diagnosis The other form is a diffuse luetic myocarditis This is also quite rare and produces a rapid development of congestive heart failure that fails to respond to the customary methods of treatment Syphilitic disease of the heart muscle has been regarded by some observers as being quite common It used to be looked upon as a common *cause of angina pectoris, especially in younger people The opinion has* gradually changed and now it is thought that syphilis is a rare cause of heart disease apart from its effect on the aorta or the aortic valves

TREATMENT

When congestive failure develops from syphilitic heart disease the response to treatment is rather unsatisfactory The hope, therefore, lies entirely in the preventive aspects of this disease All the efforts that are being made to diminish the incidence of the primary infection will naturally prevent the subsequent development of this form of heart disease Likewise the early detection of syphilitic infection and the careful and thorough treatment of syphilis in its early stages will do a great deal to prevent these late complications In fact it is already apparent, since the introduction of the Wassermann test, the discovery of salvarsan and penicillin and the present public health campaigns against venereal disease, that syphilitic heart disease is becoming much less prevalent

If syphilitic cardiovascular disease is detected before either anginal or congestive symptoms have developed treatment is directed in the hope of preventing or delaying such complications In the past, a course of potassium iodide and a heavy metal such as mercury or bismuth would be given for one month and then followed by arsenic intravenously In general, this would consist of 10 to 20 drops of saturated solution of potassium iodide three times daily and 0 013 to 0 026 gm ($\frac{1}{5}$ to $\frac{2}{5}$ grain) of mercury succinimide intramuscularly three times weekly After one month, weekly injections of neosalvarsan would be given intravenously, increasing the dose gradually from 0 2 to 0 45 gm Instead of mercury or as an adjunct to it bismuth salicylate (0 2 gm in 2 0 cc of oil) might be employed intramuscularly At present a course of penicillin has become preferable Because of the rare possibility of a Herxheimer reaction one begins with a small dose and rapidly increases the amount It is too early to know

what the ideal program should be The following would seem to be a reasonable course The first day 10 000 units may be given intramuscularly six times daily at four hour intervals The dose then is doubled each day until each dose is 160 000 units, then continued at that level The entire period of treatment might extend over ten to fourteen days The total amount of penicillin would be about 7,000 000 to 10,000 000 units This program could be altered as to dosage or intervals of injections, or one might see fit under certain circumstances to give single daily injections of long acting procaine penicillin It is not the purpose to push antiluetic therapy to the point of making the Wassermann test negative Although one would welcome this reversal it often is impossible to change the reaction of the blood and continued vigorous therapy may do more harm than good After a period of treatment has been carried out as outlined above, it may be repeated once or twice a year

When congestive failure is present all the customary methods of treatment are employed such as digitalis and diuretics (Chap 20) I have seen practically no evidence that antiluetic therapy has been useful at this stage of the disease, except in a rare case of localized gumma of the left ventricle with aneurysm which promptly disappeared on treatment However, penicillin might be given in the hope of delaying further progression It is particularly inadvisable to use arsenic intravenously if the coronary arteries are involved and anginal symptoms are present for I know of two instances in which sudden and unexpected death occurred a few minutes after an intravenous injection of salvarsan

When pain is due to disease of the aorta, especially when a definite aneurysm is present, antiluetic therapy can be very efficacious The pain may disappear entirely This type of pain should be carefully distinguished from anginal pain, for intravenous therapy should be avoided in the latter condition If aneurysmal pain is very troublesome and refractory to medical treatment surgical measures might be considered

In a few isolated and properly selected cases of sacculated aneurysm of the aorta wiring has been performed apparently with some success The cases in which this operation may be applicable are those in which the heart is essentially normal and the aneurysm both large and localized The purpose is to produce a firm clot in the aneurysm and thereby prevent or retard its continued enlargement Quite recently the operation of wiring has been superseded by wrapping the aneurysm with cellophane This is more applicable to the descending arch and thoracic aorta The purpose is to prevent further dilatation or rupture and to relieve pain

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Bacterial Endocarditis

There has been considerable confusion in the minds of physicians concerning the term "endocarditis" and its clinical significance When the valves are involved by acute rheumatic fever there occurs an acute endocarditis which often leads to valvular deformities and eventually to what is called chronic rheumatic valvular disease of the heart The original endocarditis under these circumstances is nonbacterial If the heart is examined post mortem at the acute stage of the disease the valves will show only slight alterations Pinhead vegetations may be found and slight irregularities of the valve margins may be present Bacteria will be present but rarely in the blood stream or on the valves In other words, until the actual cause of rheumatic infection is discovered acute rheumatic endocarditis is regarded as a nonbacterial endocarditis Rheumatic endocarditis is therefore an affection from which clinical recovery takes place in the great majority of cases and as a result of which chronic valvular deformities develop years later which may lead to congestive failure and other complications of rheumatic valvular disease The condition which engages our *attention in this discussion, on the other hand, is a bacterial affection of* the valves which was almost always fatal before the discovery of penicillin Therefore, when discussing 'endocarditis' two forms should be considered The first form is the nonbacterial endocarditis which is mainly rheumatic The second form is bacterial endocarditis which for purposes that will become evident can be conveniently subdivided into two subgroups, acute and subacute

ACUTE BACTERIAL ENDOCARDITIS

Most of the clinical features of acute bacterial endocarditis are similar to those seen in subacute bacterial endocarditis, except that they occur more rapidly, the whole illness is more violent and it lasts a much shorter time The cause of the brevity and the fulminating nature of the disease is the type of microorganism that is concerned and the particular disease of which the endocarditis is only a part The more common organisms responsible for acute bacterial endocarditis are hemolytic streptococci, pneumococci staphylococci, gonococci and influenza bacilli There are rare instances in which other bacteria cause this same disease Occasionally an infection with one of the above organisms may run a prolonged

course and would rightly belong to the type designated as subacute This is particularly true of *Bacillus abortus* infection (undulant fever) The subacute type, however is so common and so distinct that for practical purposes it deserves to be distinguished from those designated as acute endocarditis

Acute bacterial endocarditis is a part of an overwhelming general infection with involvement of the blood stream It is always secondary to some other primary disease process The patient may have an acute or subacute gonorrheal infection and during a phase of bacteremia, if the valves of the heart become involved, acute bacterial endocarditis due to the gonococcus develops Another has an ordinary lobar pneumonia, and in a similar fashion a pneumococcus infection of the valves develops In this way almost any infectious process can at times become the primary cause of involvement of the valves

It is often difficult and at times impossible to diagnose this condition The evidence of endocardial involvement is apt to be overshadowed by the severe septicemia and underlying disease A patient is very ill with pneumonia or with streptococcus septicemia and the fact that the valves are affected may be overlooked unless significant murmurs develop or embolic phenomena occur These are not invariable, however There is present a considerable fever with or without chills, rapid pulse and the general appearance of a severe acute infection The spleen becomes enlarged, petechiae and emboli are common and meningitis is not rare Many of these features merely indicate a severe infection and do not necessarily point to a disease of the valves The finding of a positive blood culture establishes the fact that there is a septicemia but does not prove that bacteria are lodged on the valves This accounts for the fact that many such cases are first detected on postmortem examination

It is of some interest that although pneumococcus endocarditis is quite common among fatal cases of pneumonia, it cannot be regarded as a cause of chronic valvular diseases I have seen only one case in which it seemed that an attack of pneumonia was the cause of a chronic valvular disease In this instance there were no murmurs or other evidence of heart disease before the illness and definite signs of aortic insufficiency appeared directly after the pneumonia When the physician is inquiring into the past history of his patients and is told that there was some leak in the heart ever since an attack of pneumonia he has reason to doubt the assumed relationship On closer scrutiny it may be found that the murmur antedated the attack of pneumonia and was due to a previous rheumatic infection or that the alleged pneumonia was in itself rheumatic in type or actually an instance of rheumatic pericarditis with signs of compression of the lungs which cannot be distinguished from the signs of pneumonia However, in the future the situation may be different As some recoveries may now be expected following the use of the newer drugs we will undoubtedly see cases of chronic valvular disease resulting from cured instances of acute pneumococcus endocarditis

Unlike subacute bacterial endocarditis the acute form frequently affects

valves that were previously normal Both sides of the heart are vulnerable to this infection Although the aortic and mitral valves are more commonly involved, the pulmonary and tricuspid may also be affected The disease runs a course of several days to a few weeks and was formerly almost always fatal

Treatment

In the past, many chemicals and sera have been used without success With the discovery of sulfonamides some cures were obtained and with the introduction of penicillin and related drugs many more recoveries take place The actual details of this treatment will be discussed under subacute bacterial endocarditis for it is essentially the same in the two conditions Suffice it to say that because the illness may run a short and stormy course, the exact microorganism should be identified as quickly as possible and the dosage of the drug employed should be larger rather than smaller than might have been needed However, it would hardly be wise to delay treatment in a suspected case merely because a definitive diagnosis had not yet been established or the blood cultures were still sterile As a result of this point of view we may now see recovered cases with compensated chronic valvular disease, not rheumatic in origin, that we have never seen before Furthermore, at present bacterial endocarditis is being prevented as a result of the liberal use of these preparations during febrile illnesses before the valves have been involved, as well as being cured after such involvement In fact, cases are being cured that have not and could not have been diagnosed

The entire problem of treatment has changed since the discovery of the new antibacterial agents, i e, sulfonamides, penicillin and streptomycin Physicians are so prone to use these drugs for febrile illnesses, even if the exact diagnosis is not established, that no doubt many cases that formerly would have ended up in a bacterial endocarditis now recover The result is that at pre ent this disease is seen more rarely and when already present need not progress to the full blown clinical picture formerly seen Both the acute and subacute types of bacterial endocarditis are now so responsive to treatment that it is becoming rare to observe all the dramatic complications and developments that previously occurred which were so disastrous and fatal The situation is not unlike the disappearance of the ravages formerly witnessed in the course of pernicious anemia before the discovery of liver therapy

SUBACUTE BACTERIAL ENDOCARDITIS

Predisposing Causes

As a result of considerable accumulated experience it has seemed that there are certain individuals who are more prone to develop this disease and others who are much less so As a rule patients who develop subacute bacterial endocarditis have had some sort of heart murmur for years but otherwise have been fairly well The heart is only slightly or moderately

enlarged, the rhythm is regular and there is no hypertension They have had little if any dyspnea and have been able to carry on usual activities When the original infection was rheumatic fever, they are apt to have had one or only two bouts of this, thereafter remaining free from recurrent rheumatism In other words, those patients with valvular deformities who on the whole are in comparatively good health are most vulnerable In contrast to this, it is rare indeed to see the disease develop in patients who have had chronic persistent auricular fibrillation or who have previously had congestive heart failure or hypertension In fact it is not very common in outspoken cases of mitral stenosis, particularly in those in which there is marked constriction of the valve It is frequent in patients with well-compensated aortic insufficiency or stenosis or mitral insufficiency It practically never develops in a previously normal heart but rather in those which have some abnormality of the valves or endocardium, either rheumatic or congenital in origin In general it may be said that 20 to 25 per cent of all patients suffering from valvular disease formerly succumbed to bacterial endocarditis

In an analysis of 111 cases of subacute bacterial endocarditis it was found that a past history of rheumatic fever was obtained in forty-two instances of chorea in three, and of both in six cases There were eleven other patients who had a history of scarlet fever There is an indirect association between scarlet fever and rheumatic fever in that the latter may follow in the wake of the former Some patients who develop a heart murmur after an attack of scarlet fever will be found on close scrutiny to have had mild limb or joint pains during their convalescence and it can be surmised that they actually had an attack of mild rheumatic fever which was precipitated by the scarlatina infection Recently it has been found that those children who had cardiac involvement supposedly as a result of an attack of scarlet fever have had the same high familial incidence of rheumatic heart disease as occurred in ordinary cases of rheumatic fever This leads one to believe that scarlet fever produces a chronic endocarditis only in those susceptible individuals who are already potentially rheumatic Of the entire group of 111 cases, in only one could no other cause but a luetic aortic insufficiency be found to account for the previous valvular injury There were eight other cases in which there was a positive Wassermann reaction in the blood but in all there was stenosis of the aortic or mitral valve or a past history of rheumatic fever which made it likely that a rheumatic rather than a syphilitic lesion was the predisposing cause Congenital lesions were the site of the bacterial endocarditis in five cases

Bacterial endocarditis is rather rare during pregnancy but is more likely to develop during the first few months after delivery in those women who have the appropriate cardiac background This may be due to the increased amount of dental work done in these months or to the presence of bacteria in the uterus and vagina Streptococci have been found in the uterus in many cases for some days after delivery Prophylactic penicillin would therefore be advisable for several days after the beginning of labor

The time elapsing between the original injury to the valve and the development of bacterial heart disease is difficult to ascertain accurately In a group of sixty cases the average interval between the first time the patients knew they had ' heart disease and this final disease was about twelve years The shortest interval was one year and the longest was forty-five years

Incidence

Males predominate over females in the proportion of three to two despite the fact that among all cases of mitral stenosis the proportion is two to one in favor of females This reflects the relative antagonism between mitral stenosis and subacute bacterial endocarditis The most prevalent decade is 20 to 29, although the disease occurs in individuals at all ages from childhood to old age It is curious that although males strongly predominate after the age of 30 the reverse is true under the age of 20

Clinical Features

Subacute bacterial endocarditis or endocarditis lenta is a fairly common condition and is mainly due to the *Streptococcus viridans* The infection becomes implanted almost invariably on previously injured valves Generally there has been some rheumatic valvulitis from which a satisfactory recovery had taken place or much less frequently there has been some congenital defect like patent ductus arteriosus, ventricular septal defect or bicuspid aortic valves Even when evidence of an early rheumatic infection cannot be elicited it will be found that some sort of heart murmur was present before this final infection developed

The onset of the disease is rather gradual It generally is initiated by a so-called "simple cold or sore throat or not infrequently by the extraction of a tooth or the removal of tonsils Rarely a local septic infection such as is seen after a simple abrasion of the skin may be the precipitating cause Bacterial endocarditis due to *B coli* and staphylococcus has occurred following urethral manipulation such as the passing of a sound It may occur post partum or postoperatively if there has been some minor secondary infection When these causes usher in the disease it must be borne in mind that if the heart were originally in a normal state subacute bacterial endocarditis would less likely have developed It is because the patient already has some old abnormality of the endocardium, especially if he belongs to the vulnerable group, that a few stray bacteria in the blood stream start growing on the valves Very frequently the onset is griplike or may resemble typhoid fever The patient may have complained of a cold and state that since then he has not felt well Malaise, anorexia, sweats and chills gradually develop There is a slow loss of strength and weight, although during the early few weeks many are able to do and some actually continue at their usual work Fever is practically always present and generally is of the swinging type, about 98 to 99 F in the morning and 101 to 103° F in the evening Occasionally the fever is very slight, hardly rising above 99 F for some time

Although many of the clinical features commonly seen in previous years are now often lacking because of early treatment, it is helpful to describe them in some detail as they may appear in an untreated case They will still occur in neglected cases or in those that do not respond to therapy The various important features of this disease occur in no constant order In some, only the symptoms listed above develop for a long time in others, complications that ordinarily come late may be the initial event calling attention to the gravity of the situation I recall an instance in which a young dentist suddenly developed blindness in one eye due to a retinal embolus Although he had not felt exactly well for a week or two, he was at work when the visual accident occurred The significant points to bear in mind are petechiae, splenic enlargement, red blood cells in the urine, clubbing of the fingers, painful finger tips, emboli and a positive blood culture These are particularly important because they help to distinguish this disease from an active rheumatic infection of the heart or its valves

The petechiae are small oval hemorrhagic areas about 1 to 2 mm in length which on close inspection will show a gray or white center They are commonly found in the conjunctival sac, the mucous membrane of the mouth or on each side of the neck, although they may occur anywhere over the body They are almost pathognomonic of a bacterial endocarditis The spleen gradually becomes enlarged so that at first the change may be suspected by percussion and finally by palpation The enlargement is due both to embolic infarctions and to the general septic process A palpable spleen is rarely found in rheumatic fever itself or in valvular disease of the heart even when there is congestive heart failure The liver often becomes markedly enlarged during heart failure, but not so the spleen When the abdominal viscera show evidence of passive congestion splenic enlargement only occasionally reaches the point at which this organ becomes palpable on abdominal examination

The finding of a considerable number of red blood cells in the urine is also distinctive because it does not occur in ordinary heart disease unless there is an active nephritis as well, and only very rarely during simple rheumatic fever Acute pericarditis is common with rheumatic infections and very rare with bacterial endocarditis Clubbing of the fingers is common after the first few weeks of this disease It is not seen in rheumatic fever or in ordinary heart failure It does occur in congenital heart disease when there is chronic cyanosis, in chronic pulmonary infections and in some other cardiac states associated with cyanosis Its presence as a constitutional or hereditary condition at times makes its interpretation difficult Apart from the clubbing there is also a peculiar type of pain in the finger tips that deserves attention Patients often complain of sudden pain in the balls of their fingers It comes lasts a few days and then completely disappears During this time one may see a somewhat purplish spot at the tip of the fingers or under the nails which gradually fades entirely Because of the unusual location, pains of this type are highly distinctive of a bacterial endocarditis Similar pains and discoloration of

the skin can occur elsewhere, of course, but then their interpretation is not so simple, although splinter hemorrhages anywhere in the skin are fairly distinctive

The recovery of bacteria from the blood stream by cultural methods is the most important evidence of bacterial endocarditis The ease with which this is obtained varies considerably in different cases and does not necessarily depend on the height of the fever At times repeated blood cultures may be negative although other aspects of the case indicate a severe infection, whereas in some instances in which the afternoon temperature is less than 100 F a positive culture is readily obtained The organism that is found is generally the *Streptococcus viridans* or the non-hemolytic green producing streptococcus These bacteria may be slow to grow in artificial media so that the cultures should be saved for a week or two A positive blood culture is very rare during rheumatic fever and will be found in most cases of subacute bacterial endocarditis if repeated search is made

Finally one of the characteristic features of the disease is the occurrence of emboli They are common in the spleen, producing acute sharp pain over the lower lateral aspect of the left chest, in the kidney causing pain in the loin or in the abdomen with radiation to the groins, in the limbs, or in the brain with the necessary consequent complications Emboli to the intestines sufficient to produce significant symptoms are rare, although occasionally gangrene of the intestines may result The tissues involved in these various infarctions rarely break down or suppurate When the vegetations involve the right side of the heart pulmonary emboli occur

In a small group of cases, as the disease progresses, the active stage, as expressed by fever and chills, quiets down and a picture of chronic progressive renal insufficiency develops These patients appear to have chronic nephritis with nitrogen retention and marked anemia They may have little or no fever for long periods of time, show no bacteria in the blood stream and die a renal death The clinical diagnosis will then have to rest on the presence of cardiac murmurs, clubbed fingers, anemia and other general peculiarities of the illness

Rarely one may see what can be called "silent bacterial endocarditis I recall overlooking such a case which I regarded as having either neuro circulatory asthenia or mild rheumatic fever This young soldier had a mitral systolic murmur from a previous rheumatic bout and complained of weakness and mild atypical discomforts in the precordium There was no dyspnea or evidence of cardiac inefficiency For many weeks there was no fever, leukocytosis or increased sedimentation rate No clubbing, hematuria or embolic phenomena developed Only during the last days of his life was it apparent that he had any infection, when he rapidly went downhill and died At autopsy bacterial endocarditis of the mitral valve was found

There is a type of case in which the vegetations may be confined to the right side of the heart and in which peripheral arterial emboli are striking

by their absence and the blood cultures prove negative for most of the course of the illness Small emboli, however, become dislodged and produce multiple pulmonary infarctions Such cases may resemble pneumonia and be wrongly diagnosed as such This state of affairs is apt to occur when a bacterial endocarditis develops on the defect of a patent ventricular septum or patent ductus arteriosus I have seen an instance in which on postmortem examination a ring of vegetations encircled the aperture of the septum on the right ventricle while there were no vegetations in the left ventricle It seemed that the flow of blood from left to right ventricle prevented the growth of the bacteria against the stream The presenting symptoms in this case were pulmonary, and the peripheral arterial phenomena were lacking

The course of the illness, if untreated, is a gradual downhill one It *may be suddenly altered or terminated* by *peculiar accidents such as rupture* of a valve, the ventricular septum or the ventricular wall, or by a gross cerebral embolus There is even a recorded instance in which an embolus into the coronary artery from a vegetation of the aortic valve occurred with instant death in an individual who was well enough to be playing golf when he was stricken Ordinarily however, the disease lasts several months or longer with slow wasting such as one might expect with a prolonged septic process The symptomatic complaints may be very few for long periods of time In fact some would hardly think there was anything wrong and wonder why they are regarded as sick and kept in bed During the early weeks or even for months there may be no dyspnea or evidence of cardiac failure These do occur later in the disease and heart failure may be regarded as the final cause of death in a considerable portion of cases Other patients merely waste away with increasing anemia In a small number a major embolus brings to a close this lingering disease In a very few the end result is a glomerular nephritis with uremic manifestations

The Valves Involved

It is frequent to find on postmortem examination that the vegetations of subacute bacterial endocarditis involve more than one valve When they are confined to one valve they are almost as common on the mitral as on the aortic leaflets Much more rarely are the pulmonary or tricuspid valves affected A correlation of postmortem findings with the presence of heart murmurs enables one to predict fairly accurately the location of the vegetative process When the original defect is a congenital lesion the vegetations will be centered about that area involved In acquired heart disease (almost always rheumatic and only rarely luetic), if there is an apical systolic murmur and no diastolic murmur the vegetations will be confined to the mitral valve Occasionally there is only a systolic murmur but one that is best heard at the base of the heart due to aortic stenosis without evidence of aortic insufficiency Under these circumstances there will be vegetations on the aortic valve When an aortic diastolic murmur

is heard there will always be found an active bacterial endocarditis of this valve either alone or with extensions on the mitral valve When there are typical signs of mitral stenosis without evidence of aortic insufficiency the growths will be found on the mitral valve, but when an aortic diastolic murmur is also present it will be difficult to draw any conclusion for the murmur at the apex may prove to be an Austin Flint phenomenon In this way one can fairly well predict the location of the vegetative process

Differential Diagnosis

During the early days this disease offers considerable difficulty in diagnosis The insidious onset with vague nonlocalizing symptoms may lead to a diagnosis of grip Later as the fever continues and sweats develop tuberculosis will be suspected or because of spots on the skin and enlarged spleen typhoid fever will be considered More frequently because of the presence of a heart murmur, fever and some vague pains, the condition will be regarded as rheumatic fever The absence of any murmur is a very reliable clue in eliminating the diagnosis of subacute bacterial endocarditis I have seen only two instances of this disease in which either a systolic or diastolic murmur was not heard on careful auscultation Generally the murmur is of more than very slight intensity and in most cases *does not change over long periods of time The sudden development of a* new murmur or the accentuation of an old murmur occurs but rarely This indirect method of ruling out the diagnosis has proved helpful on several occasions I recall two instances in which the diagnosis of subacute bacterial endocarditis was made by competent physicians on the basis of some of the clinical features discussed above In the absence of any murmurs I expressed the definite opinion that some other cause would be found for the fever and sweats In a few days tubercle bacilli were discovered in the sputum

The presence of a murmur, on the other hand, does not necessarily mean that there is an active bacterial endocarditis It obviously is a common finding in rheumatic heart disease, and the new infection with fever, sweats and various aches and pains may be a recurrent rheumatic bout with or without rheumatic carditis This differentiation between rheumatic fever and subacute bacterial endocarditis is sometimes very difficult and always most important No matter how ill the patient suffering from the former condition may be we are hopeful of a recovery, and no matter how well one with the latter condition may appear we fear a fatal outcome unless treated vigorously with specific drugs Apart from the specific and more characteristic features previously discussed there are some more general differences between the two diseases The heart rate is apt to be more rapid in relation to the degree of fever in rheumatic than in the bacterial infection Although both may show a moderate degree of anemia it becomes eventually more marked in the latter condition Although both show a slight leukocytosis this may be absent in the presence of fever more often in subacute bacterial endocarditis Salicylates do not alter the fever or the symptoms in the one and generally do so in the other Peri-

carditis, arrhythmias and alterations in the electrocardiograms (especially prolonged P R time) are common in rheumatic and rare in bacterial infections

The presence of a distinct murmur and its proper interpretation, however, may be the main clue to the diagnosis I recall an instance when a man was taken while at work with severe pain in the left loin radiating to the genitals He also noticed grossly bloody urine It seemed like a definite case of stone in the left kidney or ureter and was so regarded by all the physicians who saw him There was a moderately loud apical systolic murmur which he had known about for many years On close questioning I learned that while at work he had not felt really well for several weeks Although there was no fever at this time, to explain both the murmur and the sudden pain in the kidney, a diagnosis of subacute bacterial endocarditis with left renal embolus was made This was confirmed by the finding of *Strepto occus viridans* in the blood stream, although the culture was made when the patient s temperature was only 99 2 F This eventuality is by no means rare in patients who have had a so called benign systolic murmur for many years

Finally, skin tests may help to differentiate rheumatic fever from bac terial endocarditis In 80 to 90 per cent of cases of rheumatic fever, if dead cultures of *Streptococcus hemolyticus* or *Streptococcus viridans* (0 1 to 0 01 cc) or the split protein products of these germs are injected intracutaneously, a positive reaction consisting of a definite wheal will result in twenty four hours In a fairly large series of cases of subacute bacterial endocarditis such tests have proved entirely negative except in rare instances This is a simple test and the reading can be made in twenty-four hours With our present experiences if the skin test is positive, one may incline to the opinion that the condition is not a bacterial endocarditis If it is negative one may suspect this diagnosis but it must be remembered that in about 50 per cent of normal individuals and 10 per cent of rheumatic patients the reaction is also negative These differences in skin reac tions and other suggestive clinical evidences of a certain incompatibility between the rheumatic state and bacterial endocarditis have led me to think that those individuals who lose their rheumatic predisposition or allergic type of response are the ones who become more susceptible to the development of bacterial endocarditis The more immune they become to the one, the more susceptible to the other It is known that patients who already are suffering from *Streptococcus viridans* endocarditis are highly immune to streptococci This is evident from the skin test and from the presence of immune bodies in their blood In fact I have injected living virulent streptococcus in considerable quantities, subcutaneously, in such patients without producing any local reaction or only a very slight one The simultaneous presence of rheumatic carditis and bacterial endo carditis on postmortem examination in a few cases is not in conflict with the view that there is an antagonism between the two diseases The bacterial infection, just like any infection, may stir up rheumatic fever and the bacterial infection may have preceded rather than followed the rheu

matism In any event, the skin test has proved of some value in differentiating doubtful cases

It is generally regarded that emboli and a positive blood culture are the two outstanding and determining evidences of a bacterial endocarditis This really needs some qualifications Emboli occur in rheumatic and other types of heart disease in which there is no vegetative endocarditis In such cases, sterile mural thrombi are generally present in the auricles, from which bits may become dislodged producing peripheral emboli either into the systemic circulation or to the lungs When such emboli occur, recovery is likely to take place Likewise, occasionally streptococci may be found in the blood stream by cultural studies when there is no reason to believe they are coming from the valves of the heart It follows, therefore, that although these points are extremely important the rest of the clinical picture is essential to validate the diagnosis of subacute bacterial endocarditis Because of the grave prognosis that the diagnosis of subacute bacterial endocarditis entails, most careful consideration must be given to other possibilities like tuberculosis, undulant fever, rheumatic heart disease and the like

Prognosis

This used to be one of the most fatal of the common types of cardiovascular disease Its duration varies considerably from a few months to a year or more Recoveries, however, have been very rare in the past, if the diagnosis is reserved for those cases that display the complete clinical picture Very likely in mild cases in which the fever does not rise above 101 F, in which one might hesitate to make a positive diagnosis, spontaneous recovery does take place On the other hand, if a fatal outcome is regarded as essential to the diagnosis there obviously can be no recoveries There have been instances of spontaneous satisfactory recovery in which a careful review of the data forces one to accept the diagnosis It is curious that on very rare occasions a patient may recover symptomatically and yet continue to harbor the streptococci in the blood stream for a long time

Treatment

When sulfonamides came into general use the first real therapeutic advance took place The mortality fell from 99 or 100 per cent to about 90 per cent Definite recoveries resulted, especially with preparations like sulfadiazine when combined with fever therapy The entire outlook changed dramatically with the discovery of penicillin and other drugs like streptomycin and aureomycin Now one can expect an overall recovery rate of 75 per cent or more The results will depend on how early treatment is instituted and how carefully the exact therapy is chosen for the particular microorganism involved There have been reports of small series of cases numbering twenty or more in which no fatalities at all occurred

A search of the older literature and a glance at the earlier editions of this book will quickly reveal an amazing variety of therapeutic procedures

that were formerly tried in the hope of obtaining cures Various chemicals, dyes, vaccines, sera, transfusions and other more fanciful procedures were employed None of them met with any success Formerly there was no advantage, except an academic one, in making an early diagnosis of bacterial endocarditis, for treatment was then no more effective than if it was made late in the course of the disease Now it is imperative to make as early a diagnosis as possible, for if too long delayed not only may cures be less attainable but irreparable damage such as congestive failure or hemiplegia may result

Once the clinical diagnosis is suspected the patient should be sent to a hospital The care, both diagnostic and therapeutic, of such a case is too involved to be carried out adequately at home Chemotherapy should not be instituted immediately, not until one and preferably several blood cultures have been taken Inasmuch as the contemplated diagnosis involves a long and expensive course of treatment it is very desirable to be on certain ground and especially to determine what microorganism is the cause of the infection There will be times that much delay may be disastrous This may not permit more than one or two blood cultures being taken If a physician has already given chemotherapy for a few days when the diagnosis is still obscure, it may render blood culture studies unreliable for some while thereafter Inasmuch as at this time the case is in its early stage, one may properly hesitate to embark on a long course of treatment and prefer to await developments for a short while In the long run much confusion and trouble may be avoided at times, by patience and intelligent observation

Another advantage of blood cultures is that when the organism is identified it will be possible to choose the appropriate type of chemotherapy and its dosage Whenever possible the laboratory should determine the concentration of the antibiotic required to inhibit the growth of the specific germ Likewise, the concentration of the drug in the blood stream on a particular course of treatment can be measured In that way the physician can feel more certain that the infection at hand will be controlled, that the dose needs to be increased or the type of drug changed At times even frequent blood cultures prove to be negative, but if there is sufficient clinical evidence to render the diagnosis likely or if there is a fair suspicion of it, a regular course of treatment should be carried out

In most cases of bacterial endocarditis, acute or subacute, penicillin is the drug of choice at present There are differences of opinion as to the dosage and the method of administration No doubt in future years the accepted procedures will change At one time constant intravenous drip was used Others employed repeated intramuscular injections of penicillin in Pitkin menstruum Still others tried continuous intramuscular injection The method in most common use and the one I employ is repeated intramuscular injections of penicillin at three or four hour intervals throughout the day The dose will vary, but a satisfactory one to start with in the average adult is 1,000 000 to 2 000,000 units daily In many cases it will become apparent in a few days that therapy is effective The

fever will quickly subside, the general well being and appearance of the patient will improve and blood cultures will become sterile The latter, though comforting, is not altogether reliable, because under chemotherapy the cultures may be negative even while the infection remains active A positive blood culture is more decisive in indicating that the treatment is not effective It is very rare for bacterial resistance to develop during active treatment, particularly when the infection is due to the alpha hemolytic streptococcus Development of resistance to penicillin of enterococci during therapy is not uncommon If the clinical progress is favorable the same dose is continued for a minimum of four weeks and possibly one or two weeks longer If the cultures remain positive or fever and symptoms persist, the dose probably needs to be increased or the drug changed

Now, as these preparations have become less expensive, it is wiser to err on the side of excessive rather than inadequate amounts Furthermore, laboratory studies may show that a sufficient concentration of the drug in the blood stream has not been reached It has seemed that some cases of well-compensated free aortic insufficiency with a hyperactive heart beat have required larger doses than would be expected One wonders whether under such circumstances, with a rapid velocity of blood flow, more of the drug is washed out of the blood stream through the kidneys Some have advised continuing treatment for six to eight weeks, even if everything appears favorable It is difficult to be dogmatic about this Practical considerations will inevitably influence the decision, such as the cost of medication, the expense of hospitalization and the discomfort of the patient from such prolonged intramuscular injections Others have felt that similar results may be obtained by giving the penicillin three times daily rather than six or eight times The suggestion that simultaneous administration of heparin is valuable has not generally been accepted I not only do not advise it but fear that it adds further risks

After treatment has been carried out for four or six weeks and the condition is apparently satisfactory penicillin is omitted If all goes well nothing untoward develops Further blood cultures should remain sterile and fever should not return Occasionally embolic phenomena occur during these early subsequent weeks which are worrisome They do not always indicate that the infection is still active At times even without further treatment all goes well and we conclude that they were sterile emboli The streptococcus skin test which generally was negative in the early stages of the disease often becomes positive as recovery takes place In fact, it is supporting evidence that the infection has been controlled if this change in skin reaction takes place Occasionally a bout of fever occurs during the penicillin therapy that is due to the drug itself It may be difficult to differentiate this from fever due to the underlying disease

In the case progressing favorably the temperature will remain normal, the blood culture negative, the white blood count and sedimentation rate will be normal and the patient will give evidence of a sense of well being If the clinical and laboratory features point to a persistence of the infec-

tion further treatment is necessary At this point the dose of penicillin may need to be doubled or quadrupled If the original organism was gram-negative or as occasionally happens if a mixed infection occurs or a second microorganism becomes involved, streptomycin, about 0 5 to 1 0 gm four times daily intramuscularly or aureomycin 0 5 to 1 0 gm four times daily by mouth would be indicated Sometimes it is appropriate to administer more than one of these antibiotics simultaneously

Methods have been used to increase the blood level of penicillin For this purpose benzoic acid, 2 0 gm four times daily, has been employed Another and more preferable drug is caronamide 2 0 gm four times daily These procedures are generally not necessary and may upset the patient s digestive tract However, at times they may be useful adjuncts to penicillin therapy, especially when one is forced to give very large doses

The most common offending organism in this condition is the alpha hemolytic streptococcus (viridans) Other organisms isolated in different cases are the enterococcus, *Staphylococcus albus*, gamma streptococcus, pneumococcus brucella organisms, *B coli* group and para influenza

Throughout this period of treatment there is very little else in the way of specific medication that is necessary The patient may be kept in bed during the early days but if he appears fairly well and essentially symptomless, as many do, he may be allowed out of bed during the latter part of this period There are no restrictions in diet, with the possible exception of salt if there is any fear of congestive heart failure If the latter is present treatment is given as one would with any case of heart failure In a few cases heart failure is present as a result of the prolonged infection before chemotherapy is effective It also may appear during the early weeks or months after the infection has been controlled It appears that in some cases the actual endocarditis precipitates a reactivation of rheumatic fever In other cases the prolonged illness, the possible increase in the mechanical deformity of the valves or injury to the coronary arterial tree by small emboli results in a deterioration of myocardial function and definite congestive heart failure

It is wise to investigate the condition of the teeth while the patient is under active penicillin therapy X-rays should be taken and if any infected teeth are found or even if it is suspected that a tooth may need to be removed at a later time it is best to do all the operative work then Because of the risk of reinfection following tooth extractions there is no better time to do the necessary operative work on the teeth than while the patient is on full doses of penicillin

Prevention

Now that we have effective chemotherapy and know fairly well which patients are most likely to develop bacterial endocarditis, preventive measures should be taken whenever possible It has been found that in about 20 per cent of cases subacute bacterial endocarditis was observed to follow within one to several weeks after a tooth extraction There is ample bacterial evidence to show that in a considerable proportion of normal

individuals the simple extraction of a tooth is immediately followed by a bacteremia, generally due to alpha nonhemolytic streptococcus Similar, but less frequent, bacteremia has been found after other dental manipulation such as filling a cavity or prophylactic cleaning of the teeth Unfortunately the same bacteremia has occasionally followed massage of the gums or the ordinary act of chewing Therefore it is imperative to advise all patients who have rheumatic valvular disease or congenital heart disease to receive prophylactic penicillin treatment

It is not certain as yet what routine to adopt for this purpose Because of the many thousands of dental extractions and the fact that the condition one is trying to prevent occurs only rarely when no precautions are taken, the entire program of prevention will break down if the procedure is too complicated and expensive At first I used to advise 300,000 units of penicillin in beeswax intramuscularly about one hour before any dental manipulation However, I have already seen a patient who followed this direction and even had a second injection several hours after tooth extraction and yet developed subacute bacterial endocarditis, which fortunately was cured by ordinary penicillin therapy At present I advise an intramuscular injection of 300,000 units of procaine penicillin G and 100,000 units of sodium penicillin twenty-four hours before, one hour before and twenty-four hours after any tooth extraction or dental manipulation This prophylaxis is not limited to the extraction of teeth, as I have recently had a case that developed subacute bacterial endocarditis after a simple filling of a tooth The above program may need revision Oral tablets, 200,000 units four times daily before and a few days after dental work may be preferable Similar preventive measures are indicated when vulnerable individuals undergo operations that entail the possibility of spreading infections, such as lancing a boil, tonsillectomy, appendectomy or termination of pregnancy The prompt treatment of sore throats and "colds with penicillin will also help to prevent vulnerable individuals from developing bacterial endocarditis

It is premature to tell what the ultimate outcome will be in treated cases of bacterial endocarditis The immediate mortality should be not more than 25 per cent This figure can be improved if cases are recognized early and treated adequately The main causes of death after recovery from the bacterial infection are cerebral embolism, congestive failure or renal insufficiency The great majority of recurrences of infection (80 per cent) appear within the first four weeks after treatment has been stopped Generally, bacterial evidence of this appears before clinical manifestation It therefore is wise to perform weekly blood cultures for a while after cessation of specific treatment If positive cultures are obtained further treatment is indicated

When the patient has done well and convalescence has progressed favorably he should gradually resume whatever duties his cardiac status will permit Many appear to be quite fit and about as well as they were before and able to perform essentially normal duties I have had a patient who has since gone through two pregnancies without mishap Others,

although in satisfactory health, have not been quite so well as before the infection It is reasonable to suspect that the valvular deformity may be greater after than before the infection Furthermore, all the circumstances that predisposed the patient to the original bacterial endocarditis remain, so that recurrences of the same type of disease in the future are not unlikely So far I have not seen an instance of a second bout after a satisfactory recovery from a first attack. With more extensive experience we may learn that recovery from this infection entails a change in the heart that may make him less vulnerable to subsequent reinfections In an appreciable minority of cases progressive congestive failure has developed during the first year after recovery

II

Congenital Heart Disease

GENERAL CONSIDERATIONS

Congenital defects of the heart are by no means rare in infants, but because many such abnormalities are not compatible with a normal development, the incidence in the general population with increasing years diminishes very rapidly Probably not more than 1 or 2 per cent of all those having organic heart disease over the age of 12 years are suffering from congenital abnormalities This figure will naturally be much greater for children under 12 years of age Until a few years ago there was very little known as to the fundamental cause of these defects except that there is some hereditary factor Recently it has been shown by the studies of Australian physicians that an attack of German measles in the mother during the first three months of pregnancy may cause certain forms of congenital heart disease, especially septal defects, in the child Congenital abnormalities of other organs such as the eyes and feet may also come about in this way The question now arises whether other virus infections of the mother may also act in this unhappy fashion

There is a definite, but not very prominent, hereditary factor in congenital heart disease In the great majority of cases the parents of children with congenital heart disease are normal However, there are enough instances where the reverse is true to make one believe in a familial tendency The following reported experiences are significant A father and son both had right aortic arch A mother and fetus had Roger s disease Three members of one family had patent ductus arteriosus It would be difficult to regard these experiences as accidental

During the first days, weeks or months of life the diagnosis of the exact type of defect is extremely difficult and often impossible to make The abnormalities of the heart are much more often multiple than single and the physical signs are not sufficiently distinctive As months and years go on and the child survives, the clinical picture becomes more definite so that in older children combinations of physical findings occur that lend themselves to more accurate analysis The result is that in those over 12 years of age it is often possible to make a correct diagnosis of at least the major defect, if there is more than one, and not infrequently of all the congenital abnormalities

Inasmuch as some of the evidence of congenital heart disease may be apparent at birth or shortly afterward, physicians should always note the presence or absence of heart murmurs or cyanosis when a child is born Such examinations should be repeated a few weeks and a few months after birth as the signs may not be detectable immediately after birth It is more difficult but also important to elicit evidence of cardiac enlargement during the first few months of life If proper notations are made at this time much confusion that often arises in later life as to whether a certain murmur developed after an infection or was there from birth, will disappear

It is not the purpose of this discussion to take up the numerous rarities of congenital cardiac abnormalities Attention will be focused on several conditions that are met with in older children and in adults which are common enough and sufficiently discrete to merit emphasis

It is well known that cyanosis is often a striking feature in congenital heart disease This cyanosis has a different mechanism than that which is seen in acquired heart disease In the former the cyanosis is due to venous blood reaching the arterial blood in the chambers of the left heart or aorta without going through the lungs It indicates a right to left (venous-arterial) shunt through one of the septa of the heart or a malformation of the large vessels (aorta, pulmonary artery or vena cava) If there is a defect in the auricular or ventricular septum, some of the venous blood may pour into the left auricle or ventricle from the right chambers and become admixed with the arterial blood This then is expelled through the aorta to the general circulation and if there is a sufficient amount of unaerated venous blood, cyanosis will be present Whether cyanosis does develop in cases of a septal defect will depend on the size of the aperture and the pressure relations in the two sides of the heart Inasmuch as the systolic pressure in the left ventricle is greater than in the right, the flow of blood through a ventricular septal defect will be from left to right and there need be no cyanosis If however, the septal defect is great there is an appreciable flow and admixture of blood from right to left during the diastolic filling of the ventricle when the intraventricular pressure is nil or very slight Furthermore, if for any reason the pressure in the right side becomes greater, as during violent coughing, or the pressure in the left side falls, as in left ventricular failure the balance may favor a flow of blood from the right to the left and one may see cyanosis develop Finally, venous blood may be expelled into the peripheral circulation because the aorta comes off completely or partly from the right ventricle Therefore it must be clear that cyanosis in congenital heart disease does not arise because of congestion in the lungs with a slow pulmonary circulation and the resultant insufficient oxygenation of the blood as occurs to some extent in ordinary congestive heart failure, or because of peripheral venous stasis, but is due to the failure of some of the venous blood ever to reach the lungs When heart failure develops in patients with congenital cardiac disease both mechanisms may be at work

There is another mechanism involved in the production of cyanosis in

congenital heart disease recently investigated by Cournand, Dexter and others They have found that in many cases associated with increased pressure in the pulmonary artery (atrial septal defect, patent ductus and Eisenmenger's syndrome) progressive disease of the finer branches of the pulmonary artery may develop This may be the cause of part of the cyanosis present in these cases, just as occurs in chronic cor pulmonale If patients with cyanosis breathe 100 per cent oxygen and there is no change in the arterial oxygen content, the cyanosis may be regarded as entirely due to congenital right to left shunt If the arterial oxygen increases, the inference is that improved diffusion took place in the lungs, and a certain part of the cyanosis was due to pulmonary pathology If the arterial oxygenation rises to normal, the cyanosis could not be due to right to left shunt at all, as that defect would have been unchanged and must have resulted from the pulmonary alteration This, of course, does not apply to cases like methemoglobinemia

It is of some interest to note at this point that cyanosis has been con fused with argyria I have seen a considerable number of instances in which patients were treated for heart disease because of prolonged "cy-anosis,' in whom the discoloration of the skin was the result of long continued use of silver-containing nose drops One was a boy 9 years old who had been regarded as a congenital cardiac since infancy The mother had instilled nose drops once or twice a day since the age of 3 months because of a 'rhinitis and sinusitis The skin had the typical slatelike discoloration of argyria The differential diagnosis was quite simple Apart from the fact that the heart was not enlarged and showed no murmurs, the tongue, the mucous membranes of the mouth and the conjunctivae were all normal in appearance In a congenital cardiac with such a degree of discoloration of the skin these tissues would also have shown marked cyanosis Another case was that of an adult who had also used nose drops for many years and because of the supposed cyanosis was given digitalis and treated for some bizarre form of heart disease

Confusion may rarely arise between methemoglobinemia and congenital heart disease I once saw a young man in the early twenties who had been markedly cyanosed since birth Although he had fair health he gradually developed weakness and dyspnea on effort, especially on climbing stairs Physical examination including that of the heart revealed no abnormal-ities Cyanosis was very striking and of the kind typically seen in classical tetralogy of Fallot The absence of clubbing of cardiac murmurs or abnormalities demonstrated in electrocardiograms made the case very puzzling The resident physician at the Peter Bent Brigham Hospital, Dr C B Favour, suspecting the proper diagnosis, bubbled oxygen through some venous blood withdrawn from the arm It was found that instead of turning red the blood retained the blue color This pointed to the diag-nosis of methemoglobinemia which was confirmed by chemical and spec-troscopic tests Within one *hour after the intravenous injection of 1 0 mg* of methylene blue per kilogram of body weight the color of the skin was normal The patient's symptoms all quickly disappeared This extraordi-

nary cure was subsequently maintained by administering about a 0 2 gm capsule of methylene blue by mouth daily

A more common type of cyanosis in infancy that may be mistaken for congenital heart disease is methemoglobinemia that results from drinking polluted water In some rural areas blue babies ' have been seen when breast feeding was stopped and the child was put on a formula made up with water from a polluted well The cyanosis which developed from the nitrates contained in the water would quickly disappear in a few days if pure water were used Some such cases have even been treated by x-ray for assumed thymus enlargement

The second striking feature is clubbing of the fingers and toes This accompanies the prolonged cyanosis and does not come merely because there is a congenital defect One naturally ascribes the clubbing to the anoxemia in the arterial blood attendant to the cyanosis The exact relationship, however, is not clear for clubbing of the fingers is found in a variety of conditions, in many of which there is no cyanosis or anoxemia It occurs in subacute bacterial endocarditis when there is no heart failure whatever It is common in abscess of the lung when cyanosis and anoxemia are absent It does occur in other conditions with prolonged cyanosis such as chronic emphysema If there is a single cause for the clubbing it has not as yet been discovered

Furthermore, congenital heart disease is often but by no means invariably, productive of loud murmurs When such murmurs are heard in infants or children, a congenital defect must be suspected unless there is some other obvious cause such as anemia or an active rheumatic infection Pulmonary tuberculosis is a not infrequent development in those suffering from congenital heart disease It is of some interest that acquired rheumatic heart disease is fairly common among congenital cardiac patients who reach adult life Of greater importance is the fact that bacterial endocarditis is a frequent complication and cause of death in many persons with congenital abnormalities The bacteria have a predilection for any abnormality of the internal lining of the heart or great vessels Stunted or retarded bodily growth may also result from congenital heart disease when it is accompanied by an appreciably diminished outflow of blood through the aorta Finally, it is commonly associated with other congenital abnormalities and with mental retardation or idiocy

IDIOPATHIC HYPERTROPHY OF THE HEART

Occasionally idiopathic hypertrophy of the heart may be congenital Such patients merely show cardiac enlargement without any valvular or septal defects They rarely live longer than a few years and present the picture of increasing cardiac embarrassment with dyspnea The cause of the enlargement is not known but one might suspect that a careful study of the coronary circulation would reveal some defect either in the larger or the very fine branches in some of these cases In a few cases considered as congenital cardiac hypertrophy there is reason to believe that the condition was due to faulty metabolism, since extensive glycogen de-

posits were found in the heart muscle Others probably were the result of prolonged anemia or avitaminosis and possibly to paroxysmal rapid heart action, all of which can cause cardiac dilatation There is reason to suspect that if all possible known factors associated with cardiac hypertrophy could be studied there would remain very few if any of the so-called idiopathic hypertrophy of the heart

COARCTATION OF THE AORTA

A much more common congenital anomaly is coarctation of the aorta Although there are different degrees and locations for this narrowing the most common site is at the arch of the aorta just beyond the left subclavian artery at the point where the ductus arteriosus develops This ductus often remains patent in cases of coarctation, and frequently the foramen ovale is also open The constriction of the aorta leads to a definite sequence of findings, more or less extensive, depending on the degree of narrowing The ascending aorta proximal to the coarctation becomes dilated and although there may be a slight dilation of the aorta for a short way below the constriction, beyond this the thoracic and abdominal aorta is much smaller than normal Extensive and enormous anastomotic arteries develop which bridge the blood stream from the first portion of the aorta proximal to the constriction to the lower part of the body The internal mammary, the intercostal and other arteries increase tremendously in size Two other concomitant abnormalities are common in cases of coarctation of the aorta, i e, bicuspid aortic valves and aneurysms of the cerebral vessels The former may become the site of bacterial endocarditis and the latter may result in cerebral hemorrhage

As a result of these abnormalities definite objective findings become manifest that enable one to make a satisfactory clinical diagnosis Not so long ago all cases of coarctation of the aorta were first detected on post-mortem examination, then later on, except on very rare occasions, the roentgenologist would first make the diagnosis, now it can readily be made by the clinician Adults with this condition develop hypertension in the arms, although the blood pressure in the legs is lower than normal Normally the pressure in the legs should be higher than in the arms Inasmuch as determinations are not made routinely of the blood pressure in the legs, this discrepancy can easily be overlooked It should become the habit of all physicians to feel for pulsations over the abdominal aorta and femoral arteries, if these pulsations are absent or markedly diminished, *especially if the patient has hypertension and if there is a basal* systolic murmur, coarctation of the aorta should be suspected and measurements of the blood pressure in the legs should be made If the pressure in the legs is less than in the arms, a presumptive diagnosis can be made A second feature is the finding of pulsating superficial arteries over the back, around the scapulas Wormlike pulsating subcutaneous arteries may be found there Examination of the heart may show some enlargement and murmurs at the base of the heart of the type seen in hypertension, aortic valvular disease or patent ductus arteriosus, i e, there may be

systolic with or without diastolic murmur The systolic murmur which is always heard at the base of the heart may be as loud or louder in the interscapular region Finally the x-ray of the chest generally can establish a conclusive diagnosis This will show a prominent ascending aorta and an absence of the aortic knob of the descending aorta A swallow of barium may identify dilation of the thoracic aorta below the constriction The x-ray occasionally may show calcified aneurysm of an intercostal artery coming off this aneurysmal dilation of the aorta It will also show a peculiar notching or scalloping of the lower margins of the ribs due to the erosion produced by the enlarged pulsating intercostal arteries These changes are pathognomonic of coarctation of the aorta and are most commonly found in the fourth to the seventh ribs They rarely appear before the age of 4 years but have been seen in an infant 9 months old

It was just mentioned that an aortic diastolic murmur may occasionally be present This may be due to a concomitant rheumatic aortic lesion, to a complicating bacterial endocarditis of the aortic valve, or to dynamic or relative aortic insufficiency The following is an example of the latter type A man 20 years old had a huge heart with signs of coarctation There was a grade III systolic and grade III aortic diastolic murmur There was no past history of rheumatic infection The blood pressure was 225/70 mm in the arms and 140/0 in the legs Postmortem examinations confirmed the diagnosis of coarctation of the aorta The heart weighed 900 gm but showed no valvular disease and no cause for the diastolic murmur

When it is appreciated that about 0 05 per cent of the entire population have this abnormality it becomes important for the physician to be familiar with it These patients often survive to adult life and may even reach old age, but the average age at death is 35 years Their difficulties are those that accompany vascular hypertension i e, myocardial failure and cerebral hemorrhage Besides this they are prone to two other peculiar complications, rupture of the aorta and bacterial endocarditis

There is no medical treatment for this condition apart from the general advice given to patients with asymptomatic hypertension and those subject to cardiovascular accidents The discovery of surgical methods designed to correct this defect has been a great advance It is now possible to resect the constricted portion of the aorta and make an end to end anastomosis In cases where the length of constricted aorta is too great for this operation a graft of another human aorta appropriately preserved, may be transplanted Gross has performed such operations with excellent results and it appears that aortic grafts of this sort will function perfectly Even the subclavian artery has been ligated to the distal end of the area of constriction to by pass the obstruction These various operations have resulted in many cures The operation is best carried out before the age of 20 years and preferably at the age of 10 to 12 years The operative mortality is now about 5 to 10 per cent The reason for early surgical treatment is that in the course of time the aorta distal to the constriction may become thin and dilated It then may not be strong enough for

surgical manipulation, or rupture may occur postoperatively when normal blood flow and normal pressure returns

DEXTROCARDIA

Congenital dextrocardia is of two types In the first type the apex of the heart points to the right but is formed by the right ventricle The heart is essentially rotated more to the right so that the left ventricle lies more anterior than normally but yet to the left of the right ventricle This condition is due to a congenital arrest in development and therefore is generally associated with other cardiac abnormalities such as absence or defects of the septa or transposition of the arterial trunks Such patients are apt to suffer serious handicaps The second type is called the 'mirror picture dextrocardia and is practically always associated with a complete transposition of the other viscera (the liver and appendix being on the left, the spleen on the right, etc) Here the apex of the heart points to the right, is felt near the right nipple but is made up of the 'left ventricle' (the one receiving blood from the lungs) The right ventricle and auricle lie to the left There are no other defects in the heart and such patients have no symptoms referable to the circulation This condition is often overlooked until accidentally found on routine examination It cannot be regarded as an arrest in the development It suggests rather an inversion of the embryo in its relation to its primitive yolk sac The electrocardiograms in mirror picture dextrocardia are absolutely pathognomonic Lead I will show inverted P and R waves just as would result if the lead wires in taking a normal Lead I were accidentally reversed (Chap 21, Figs 126, 127) Patients with this condition have a normal life expectancy and need not be restricted in their activities

/ PATENT FORAMEN OVALE AND ATRIAL SEPTAL DEFECT

Patency of the foramen ovale is probably the most common of all congenital cardiac anomalies In intrauterine life this opening in the interauricular septum permits the blood to flow from the right to the left side of the heart without going to the lungs, for the latter are compressed and functionless During the first few months the foramen ovale normally closes but in a considerable number of individuals a greater or lesser patency persists When the foramen ovale is small it produces neither signs nor symptoms of heart disease When it is large it may result in enlargement and dilatation of the right auricle and ventricle and the pulmonary artery There may or may not be any murmurs and congestive heart failure due to strain on the right side of the heart may result Paradoxical embolism may occur in this condition as in any instance in which defects in the septa of the heart are present Under such circumstances a thrombus from a peripheral vein or from the right auricle on becoming dislodged may go through the septal defect and produce an arterial embolus (to brain, kidney, spleen, etc)

There are other defects of the interauricular septum which are similar in many respects to patency of the foramen ovale The lower part of the

septum (persistent ostium primum) or the upper portion (persistent ostium secundum) may be patent The entire auricular septum may be absent, producing a cor triloculare biventriculare All these auricular defects produce similar changes, the degree of which depends to some extent on the size of the patency The right auricle and ventricle may become enormously dilated and hypertrophied and the pulmonary artery considerably enlarged while the aorta is actually smaller than normal Slight cyanosis is common and clubbing is rare These cases of atrial septal defect may show no murmurs, or there may be a slight to moderately loud basal systolic and occasionally a pulmonary diastolic murmur Both the first and second heart sound in the pulmonary area are often accentuated This may occur in other conditions in which the pulmonary artery is dilated and there is increased pulmonary pressure or flow In fact, pulsation may be felt in this area under such circumstances Right axis deviation in the electrocardiograms is common I have seen two instances in which the pulmonary artery was so dilated that it compressed the left recurrent laryngeal nerve causing paralysis of the left vocal cord The x ray will show slight, moderate or marked spherical enlargement of the heart, dilatation of the pulmonary artery and increased hilar pulsations and pulmonary vascular markings Some patients with interauricular septal defect also have mitral stenosis (Lutembacher syndrome), and they then may also have auricular fibrillation Whenever a patient is known to have mitral stenosis and fails to show a dilated left auricle, one should consider the possibility of Lutembacher syndrome It is thought that the early development of acquired rheumatic mitral stenosis with the resultant increase in left auricular pressure tend to enlarge the foramen ovale which was already present in these cases The septal defect acts as a release valve and often enables such patients to carry on fairly well In general, patients with atrial septal defect almost never develop bacterial endocarditis and may live fairly long and useful lives Although a few attempts have been made to operate on auricular septal defects, it is too early to judge their value

PATENT VENTRICULAR SEPTUM

Interventricular septal defects are quite common and rank next in frequency to auricular defects They are generally associated with other abnormalities such as right-sided position of the aorta or pulmonary stenosis, but may occasionally occur as the only lesion (Roger s disease) The patency is usually small measuring 1 to 2 cm in diameter and is most often located in the upper or membranous part of the septum just below the aortic valve This condition generally produces no symptoms of cardiac embarrassment but despite this only a few live beyond the age of 30 or 40 Probably this is because subacute bacterial endocarditis occurs in about 40 per cent of the cases Now with the aid of chemotherapy, cases may be expected to live much longer When bacterial endocarditis does occur the vegetations are apt to be located in a circular fashion around the right ventricular aspect of the defect or on the right ven-

spicuous or in an abnormal position The most distinguishing sign is the finding of left axis deviation or left ventricular enlargement in the electrocardiograms In very early infancy the tracings may show no preponderant hypertrophy of either ventricle, when normally in the first months of life electrocardiograms always show right ventricular preponderance This is the only type of congenital heart disease with marked cyanosis that shows electrocardiographic evidence of preponderant hypertrophy of the left ventricle, the electrocardiogram therefore serves as a valuable aid in differential diagnosis

The Blalock-Taussig operation is indicated in properly selected cases of this type

EISENMENGER COMPLEX

The Eisenmenger complex is a rare condition that resembles the tetralogy of Fallot It consists of a ventricular septal defect, dextraposition of the aorta, but no stenosis of the pulmonary valve although there may be pulmonary insufficiency Cyanosis is apt to come late and is slight or moderate and clubbing may be slight or absent A moderately loud systolic murmur will be present over the midprecordium, and possibly a pulmonary diastolic murmur The electrocardiogram will show right axis deviation Cardiac arrhythmias are common and the x-ray shows an enlarged heart and pulmonary artery and increase in the size of the smaller pulmonary vessels Effective surgical treatment is as yet unavailable for this condition

PULMONARY STENOSIS

Pulmonary stenosis as an isolated lesion is not so rare as was formerly thought The physical findings are a loud systolic murmur at the base of the heart, best heard in the pulmonary area, and a systolic thrill There *is also marked electrocardiographic evidence of right ventricular enlargement* In infancy and childhood the pulmonary artery is not likely to be prominent on x-ray examination, but during the second and later decades it almost always becomes considerably enlarged Pulmonary vascular markings are normal or decreased It is compatible with a fair state of health Attempts are being made to enlarge the stenotic valve by incising it through the right ventricle Some success with this operation has been obtained by Brock of London and American surgeons

PATENT DUCTUS ARTERIOSUS (BOTALLI)

During fetal life blood leaving the right ventricle is expelled through the pulmonary artery, then by way of the ductus arteriosus into the aorta and in this way to the placenta where it becomes oxygenated The blood obviously does not go to the lungs, which are compressed and atelectatic During the first week of life the ductus arteriosus, having lost its function, normally disappears when venous blood begins to flow to the lungs When this opening between the pulmonary artery and the aorta fails to close the condition called patent ductus Botalli results Although this is commonly accompanied by other congenital abnormalities it often is

present as the sole or the major defect This does not produce cyanosis or clubbing because the flow of blood is from aorta to pulmonary artery (arterial to venous) and there results no admixture of unoxygenated blood in the systemic circulation

There need be no symptoms of circulatory distress from patent ductus arteriosus and its presence is often detected accidentally on routine examination The systolic blood pressure is normal but the diastolic is low, just as occurs in aortic insufficiency or any arteriovenous fistula The main diagnostic finding is a peculiar murmur heard best in the second left intercostal space and often just to the right of the left midscapular region The murmur when typical is continuous throughout systole and diastole and has been called a machinery murmur because of its quality It may rarely last only through systole and is often accompanied by a systolic thrill in the pulmonary area The typical murmur seems to envelop the pulmonary second sound and is loudest during the latter part of systole (Fig 191) X-ray examination may also aid in the diagnosis in showing a marked prominence of the pulmonary artery which becomes dilated Other roentgenologic findings in the order of frequency are slight cardiac enlargement, slight dilatation of the left auricle, engorged pulmonary vessels and exaggerated pulsations of the left ventricle and pulmonary artery The electrocardiogram either shows normal tracings or may indicate slight left ventricular preponderance The condition is compatible with good health and is often seen in young adults In some, however, bodily development is stunted because of the diminished outflow of blood to the peripheral arteries and there may also be weakness and dyspnea on effort Pulmonary tuberculosis is not infrequent and bacterial endocarditis is a quite common complication of this defect

In 1938 the first successful ligation of a patent ductus arteriosus was reported by Gross and Hubbard Since then many patients with this anomaly have similarly been operated upon The operation is simple and effective The loud murmurs promptly disappear and the low diastolic pressure due to the shunt of blood from aorta to pulmonary artery immediately returns to normal The pulmonary artery may remain dilated and a basal systolic murmur may persist for some months after a successful operation The purposes of the operation are (1) to prevent the subsequent development of bacterial endocarditis which has been a complication in about 25 to 40 per cent of the cases of patent ductus arteriosus, (2) to prevent the retardation of mental and physical development and (3) to improve the efficiency of the circulation when there is any evidence of cardiac insufficiency The latter two objectives are obtained by stopping the considerable loss of arterial blood that leaks back into the pulmonary circuit A successful operation also will diminish the work of the over burdened left ventricle and thereby delay or prevent heart failure Needless to say, in the selection of cases the diagnosis must be certain and for the present, subjects having other congenital abnormalities should not be considered

In experienced hands the operative mortality at present is about 1 to

2 per cent and very likely will be further reduced before long Inasmuch as the average age at death of a large group of patients with patent ductus arteriosus has been found to be only 35 years, successful operations should lengthen life expectancy considerably The optimum time for surgery is in childhood, from the age of 4 to 8 years With the present low operative mortality I would advise section or ligation in all young cases, particularly if there is stunted growth, heart failure or marked pulmonary engorgement Even if there are no significant symptoms, I now advise operation in young adults in the twenties It is debatable whether individuals over 30 years of age who are doing well should be subjected to surgery The presence of cyanosis should be regarded as a contraindication to operation

The presence of bacterial endocarditis with patent ductus offers a special problem Both the congenital abnormality and the bacterial infection have been cured by surgery without the use of antibacterial medication It is amazing that in some cases blood cultures, which had been repeatedly positive up to the time of operation, became negative within minutes after division of the duct In others, cultures continued positive some days or even weeks after operation and yet finally cures were obtained It appears that once the locus for the infection was obliterated on which further vegetations could grow, the bodily defense mechanism could gradually overcome the remaining decreasing bacteremia Still other cases have been cured of the endocarditis or endarteritis by chemotherapeutic measures, particularly penicillin, but would be left with the patent ductus

At present the most satisfactory plan to follow is to treat the infection with penicillin or similar antibiotics as is done in ordinary bacterial endocarditis and to follow this in three to six months with surgery The diagnosis should be made as quickly as possible before the valves of the heart become involved by the bacteremia A spread of the vegetations from the duct itself or its mouth to the valves is by no means rare and increases the difficulty of obtaining a cure The occurrence of arterial emboli will indicate such extension to the valves or aorta, but neither such manifestations nor hematuria or splenic enlargement need dim the expectations of obtaining an ultimate cure by surgery If the patient responds satisfactorily to medication, operation may then be performed In many cases satisfactory results have been obtained, but occasionally the tissue is still so friable that disasters have occurred It now seems wiser to wait several months, when the operation will be safer However, if the infection is not responding well to chemotherapy or if there are other reasons to regard delay as precarious, operation should be performed promptly In summary, present day surgery and medicine have entirely changed the outlook of patients with patent ductus with or without bacterial endocarditis

CONGENITAL VASCULAR RINGS

There is one congenital abnormality that is of some importance because of the peculiar associated clinical findings, i e, right aortic arch Very

rarely the arch of the aorta is directed to the right and is formed by the primitive fourth right aortic arch The aorta then passes over the right bronchus to the right and behind the esophagus and trachea The pressure on the esophagus or trachea may cause difficulty in swallowing (dysphagia lusoria) or respiratory symptoms like cough and wheezing dyspnea Esophagoscopy may then reveal a constriction and the x ray examination following a barium meal will show that the arch of the aorta turns to the right and lies behind the esophagus, compressing it forward, to the right or to the left In addition there may be remnants of the congenital cords which formed the left aortic arches, thereby forming a ring around the esophagus and trachea

There are many different types of congenital abnormalities of the arch of the aorta and the main arteries that leave therefrom They mainly involve aberrant formation or direction of the subclavian artery In many cases the right subclavian artery starts from the left side of the arch of the aorta The important point to bear in mind is that if infants or children have recurrent respiratory infections or have difficulty in breathing or swallowing, the possibility of congenital vascular rings must be considered Fairly accurate diagnoses can now be made by a well trained roentgenologist even without the use of angiocardiography About 80 per cent of such cases have symptoms, and the others are symptomless Most of these cases are now amenable to surgical cure

BICUSPID VALVES

Congenital bicuspid aortic or pulmonary valves are clinically of importance only in so far as they are the frequent site upon which acute or subacute endocarditis develops Such lesions may account for some of the instances in which bacterial endocarditis develops in patients who previously showed no murmurs in the heart When bacterial endocarditis develops in patients who have not suffered previously from a rheumatic infection careful search of bicuspid semilunar valves should be made

Although there are numerous other congenital cardiac abnormalities either occurring singly or in varied combinations they are not sufficiently important to deserve discussion in this review

CATHETERIZATION STUDIES, ANGIOCARDIOGRAPHY AND SUMMARY

The introduction of the technic of catheterization of the heart and the x ray visualization of the cardiac chambers and great vessels by the use of an opaque medium (angiocardiography) have completely changed the status of diagnosis of congenital heart disease Many conditions that formerly were mystifying now can be accurately diagnosed By means of angiocardiography certain anatomic abnormalities can be visualized Catheterization studies in many ways are even more informative By measuring the pressure in the veins, right auricle, right ventricle, pulmonary artery and pulmonary capillaries, important physiologic data are obtained that frequently are diagnostic of one condition or another or

that indicate the severity of a given condition The determination of the oxygen content of samples of blood removed at various points during the procedure often affords decisive evidence that there is a left to right shunt of blood and may help to establish or to rule out various diagnoses The catheter may slip from the right to the left side of the heart, thereby giving proof of an auricular septal defect It is evident that this new information has become invaluable in the study and care of cardiac problems, especially in those suffering from congenital heart disease

Catheterization of the heart, however, is a complicated procedure requiring the cooperation of several trained workers and is quite time-consuming It might appear hazardous, but with care the risk is practically negligible In Dr Lewis Dexter s clinic, at the Peter Bent Brigham Hospital, about 1000 examinations have been carried out without any serious complication It is hoped that before long careful correlation of this information with more simple clinical or bedside data will enable the physician to do without this more elaborate method of study For the present, whenever the diagnosis is uncertain and particularly if the question of surgery is being considered, these elaborate diagnostic procedures will need to be carried out

There are some useful general considerations that are applicable in appraising congenital heart disease If cyanosis is present and the smaller pulmonary vessels appear prominent or engorged on x ray examination, a Blalock Taussig operation is not indicated However, if the pulmonary vessels are faint or less prominent than normal a shunting operation, increasing blood flow through the lungs, is likely to be helpful Whenever left axis deviation or left ventricular hypertrophy is found in a cyanosed case that may resemble tetralogy of Fallot, the diagnosis is likely to be rudimentary right ventricle with tricuspid atresia The electrocardiogram can show right ventricular hypertrophy or right bundle branch block in several types of congenital heart disease, i e, tetralogy of Fallot, atrial septal defect, Eisenmenger s syndrome and pulmonary stenosis It shows no preponderance, or there may be left axis deviation in patent ductus When right axis deviation is present with patent ductus, some additional anomaly such as a pulmonary stenosis is likely to exist Roger s disease displays no preponderance of either ventricle

The x ray is less valuable than the electrocardiogram in distinguishing hypertrophy of either ventricle It does clearly visualize enlargement of the pulmonary artery However, such enlargement occurs in a great variety of conditions such as mitral stenosis, patent ductus, pulmonary stenosis, atrial septal defect, Eisenmenger's syndrome and chronic cor pulmonale In all of these conditions except pulmonary stenosis the smaller pulmonary markings are frequently exaggerated in contrast to the appearance seen in tetralogy of Fallot, where the lungs are more bloodless and the main pulmonary artery is smaller than normal, and in pulmonary stenosis where the small vessels may be normal while the main branch is dilated

The pressure in the right ventricle is elevated in many forms of congenital and acquired heart disease In mitral stenosis and in left ventricular

failure from any cause, not only is the pressure increased in the right ventricle and main pulmonary artery but also in the 'pulmonary capillaries' This distinction helps in differential diagnosis Furthermore, in cases of chronic cor pulmonale from chronic emphysema or other forms of lung disease the pressure is elevated in the right ventricle and pulmonary artery but is essentially normal in the pulmonary capillaries Similar normal readings of pulmonary capillary pressure are obtained in most cases of congenital heart disease unless left ventricular failure is present It is clear that the determination of pressure readings coupled with the oxygen content of the blood throws considerable light on the anatomic structure of the heart and on the physiologico pathologic state of the dynamics of the heart at that time

Progress in diagnosis and treatment of congenital heart disease has been so rapid recently that literally every year brings new advances The latest development is thoracoscopy as an aid in diagnoses of congenital heart disease introduced by Fatti and Gilroy of South Africa After the production of a left pneumothorax the instrument is inserted into the chest and direct visualization of the heart and great vessels is obtained One cannot but feel optimistic of the progress that is in store in the immediate future

TREATMENT FOR CONGENITAL HEART DISEASE

There is no known effective medical treatment for congenital heart disease Digitalis is generally useless but quinidine may be helpful for cardiac irregularities present in some cases Inasmuch as pulmonary tuberculosis and bacterial endocarditis are common complications, efforts should be directed at maintaining as high a general bodily resistance to infection as possible Phlebotomy may be helpful when there is marked cyanosis and plethora and especially if there are cerebral symptoms Oxygen inhalations and morphine may be helpful for attacks of paroxysmal dyspnea Patients should be encouraged to carry on some moderate occupation whenever possible, since many are able to live a fairly long and useful life

Surgical methods are being constantly devised that have dramatically changed the outlook of many of these cases This has been particularly true of patent ductus, coarctation of the aorta vascular anomalies of the *aortic arch, pulmonary stenosis and tetralogy of Fallot* No doubt other congenital abnormalities will become amenable to surgical treatment in the near future

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Functional Heart Disease

For practical purposes it seems convenient and helpful to classify all patients having abnormal signs or symptoms of heart disease in whom there is no structural disease of the heart as suffering from functional heart disease This will naturally include a great variety of conditions In general there will be two groups of such patients, those having some abnormal physical findings in the heart and others with symptoms but without abnormal findings There will be many, of course, who will manifest both symptoms and signs There are numerous terms in use to designate these conditions and although they may connote slightly different states, they all may properly be used to specify functional heart disease in the sense described above Among the current terms are ' neurocirculatory asthenia, effort syndrome, 'disorderly action of the heart, soldier's heart," 'cardiac neurosis and nervous heart It will be seen in the succeeding paragraphs that other designations may well be used in this same group of patients such as "benign cardiac irregularity and benign systolic murmur '

Let us first consider the group of patients who have either no symptoms whatever or insignificant ones, who show abnormal signs During the course of an insurance examination or any other routine examination the physician may detect a faint systolic murmur in the absence of any other evidence of heart disease This signifies the absence of hypertension, cardiac hypertrophy, diastolic murmurs, or any other cardiac symptoms If there is no history of any previous rheumatic infection, under these circumstances, a slight systolic murmur can be disregarded and called functional or benign Such a patient, for purposes of classification, may be said to have functional heart disease (benign systolic murmur) This applies only to a faint systolic murmur

It will be found useful to estimate the intensity of systolic murmurs just as we do the amount of albumin in the urine It is also necessary to confine the term systolic murmur to a bruit that has an appreciable duration and lasts for a definite interval after the first heart sound Many mistaken diagnoses are made in designating as murmurs a prolonged first heart sound, frequently detected in hyperactive hearts and thin-chested individuals, when the interval between the first and second heart sounds is entirely clear A more detailed discussion of the systolic murmur will

be found in Chapter 17 Suffice it at this point to bear in mind that one should hesitate in considering loud murmurs as insignificant, benign or functional

Apart from the heart itself, the more important factors that seem to be responsible for systolic murmurs are anemia, hyperthyroidism, hypertension, fever, tachycardia and the emotional state of the patient In general it has been found that faint systolic murmurs, although present in only a small percentage of people, should be regarded as having no significance Those of moderate intensity may indicate some organic disease in the heart or elsewhere but also occasionally occur as a functional manifestation, and murmurs of louder intensity practically always signify some obvious organic disease

In an extensive study of systolic murmurs it did not seem to matter whether faint systolic murmurs were heard at the apex or base of the heart or whether the patient was examined in the upright or recumbent position, although the frequency of benign systolic murmurs varied somewhat under these different circumstances It is of no importance whatever that a systolic murmur, previously not present, is brought out by effort, for it was found that in practically all normal individuals systolic murmurs appeared directly after a brief brisk effort The important point in this regard is that systolic murmurs of greater than the faintest intensity deserve investigation This simple finding may be an important clue to the diagnosis of otherwise unsuspected hyperthyroidism or bacterial endocarditis Although in some cases no adequate explanation will be found for the presence of a systolic murmur and such an abnormality is consistent with a long and active life, there is a tendency at present to pay too little attention to systolic murmurs and consider them all as benign Apart from the conditions just mentioned, this type of benign systolic murmur often proves to be a manifestation of rheumatic heart disease, frequently is found to be eventually associated with stenosis of the aortic or mitral valve, occasionally is an evidence of myocardial disease and rarely of congenital heart disease Notwithstanding these considerations there remain some individuals with faint systolic murmurs who must at present be regarded as having no organic disease and in whom the diagnosis of functional heart disease (benign systolic murmur) must be made

Further physical findings frequently regarded as benign or functional are certain arrhythmias Almost all the arrhythmias that are met with in general practice can at times occur in patients who have no organic heart disease The presence of sinus arrhythmia and extrasystoles of any type is frequently the cause of apprehension and sometimes of discomfort when the condition is entirely functional The same is true of paroxysmal auricular tachycardia As our experience has broadened we have come to realize that even auricular fibrillation and flutter, paroxysmal or permanent, may occasionally be unassociated with any other disease On rare occasions ventricular tachycardia has been observed in otherwise normal individuals All these irregularities of the heart of course do occur with organic disease as well and it becomes very important to use every reason

able means to exclude organic conditions before making the diagnosis of functional heart disease (benign irregularity) It is particularly important not to overlook hyperthyroidism when auricular fibrillation is present as this may be the sole manifestation of a toxic goiter and it will be necessary to make intelligent interpretation of the basal metabolism

If any of the above irregularities are present without symptoms of angina or congestive heart failure and if other features like hypertrophy of the heart, hypertension, a diastolic murmur, significant changes in the electrocardiogram, a past history of rheumatic infection, abnormal configuration of the x-ray shadow of the heart and aorta or a positive Wassermann reaction are absent, the condition must be regarded as functional It does not follow that all these investigations are necessary to arrive at this conclusion, but these are the various points to be considered in the study of such cases in which the primary complaint is apt to be palpitation of the heart

The sensations that develop from benign extrasystoles are so peculiar and at times so distinctive that one should be familiar with them, as often the description of the symptoms given by the patient establishes the diagnosis even without any further examination They are frequently described as a 'flop of the heart, "the heart turns a somersault,' "it skips or hesitates," there is a sudden thump or jump or choking sensation in the throat, the heart suddenly sinks or a wave passes over me' Although the actual terms used are varied they are all characteristically descriptive of what is going on Some compare the sensation to 'the sudden flapping of a bird s wings' or to a "fish suddenly turning in the water The sensations produced by the extrasystoles appear to come from the premature beat itself, though they have often been thought to result from the forceful beat that follows the pause These disturbances are most apt to occur when the patient is at rest, especially while quietly seated or while trying to fall asleep This is explained either by the fact that the mind is not occupied with other things or, more likely, on the basis that while the subject is at rest the heart rate is slower and the opportunity is greater during the longer diastolic pauses for premature beats to arise At any rate the sensations are generally absent while the patient is active walking or busily engaged in his affairs, although there is one rarer type that is brought on particularly by effort

Although it has long been known that extrasystoles frequently are of no serious significance and that perfectly well individuals may have them only recently has it been possible to ascribe a definite neurogenic origin to them Beattie and Brow made animal experiments reproducing persistent ventricular extrasystoles and found that if certain nerve tracts coming from the hypothalamic region were cut the irregularity disappeared It is of some interest that the hypothalamic region is concerned with the control of our emotions They also found that if these tracts were cut beforehand the extrasystoles could not be produced by the same technic that always brought them out in animals not subjected to this treatment In other words, there is a center in the brain that can initiate or that is

intimately connected with the formation of premature heart beats This work has firmly established a structural anatomic basis for conditions that have long been regarded as functional or nervous

Treatment for patients with extrasystoles varies considerably When the extrasystoles come infrequently all that is needed is an explanation that nothing serious is going on The patient should be encouraged to carry on normal physical duties It will only rarely be necessary to inhibit the use of coffee or tobacco When the irregularity is very disturbing quinidine sulfate in doses of 0 2 to 0 3 gm two or three times daily, or even less, is often effective Papaverine (0 1 gm) administered three times a day by mouth has been advised At times the patient learns that a certain medication is effective but only necessary just before the palpitation is expected to occur

It has been stated that auricular fibrillation which generally is associated with organic heart disease can be at times a purely functional disturbance This is particularly true of the paroxysmal type of auricular fibrillation, but I believe it also occurs in a few cases when the arrhythmia is permanent In the absence of rheumatic heart disease, especially mitral stenosis, of coronary artery or hypertensive heart disease, one must always suspect that there is an underlying hyperthyroidism when auricular fibrillation, either transient or permanent is present If the basal metabolic rate is normal the possibility of a toxic thyroid gland as the cause of the auricular fibrillation is ruled out for the most part This is not invariably true for it is now believed that even some of these patients with a normal basal metabolic rate already have disease of the thyroid gland and will subsequently show an elevated rate and that the auricular fibrillation may disappear after subtotal thyroidectomy It has seemed that some such patients have been improved without surgery by taking Lugol s solution It is also interesting that most of these apparently normal patients whom I have seen with auricular fibrillation and a normal metabolic rate have looked alike, have been males and resemble in some ways patients with active hyperthyroidism

When hyperthyroidism alone is the cause of the auricular fibrillation, the irregularity may still be regarded as functional for it disappears under appropriate treatment of the thyroid gland and there remains no evidence of organic heart disease Some work has been done showing that in hyperthyroid animals transient auricular fibrillation can be reproduced by the injection of adrenalin It was found that without adrenalin such animals would never show auricular fibrillation and that adrenalin in normal animals produced only ventricular irregularities and not auricular fibrillation One might infer that in patients with hyperthyroidism the secretion from the adrenals has some relation to the production of auricular fibrillation This would explain the occurrence of transient spells of this irregularity following emotional shocks and fright At any rate it has become clear that permanent structural disease of the heart is not a necessary prerequisite for auricular fibrillation

The other general type of patients comprises those who have various

symptoms in whom abnormal physical signs like irregularities may or may not be present Such patients are often young and they complain of palpitation, weakness, giddiness, pain in the region of the heart and frequently shortness of breath The palpitation generally is associated with a normal heart rhythm They merely feel the pounding of the heart which has either a normal rate or is slightly accelerated When the rate is found to be rapid while the patient is awake it will be normal during sleep The pain is almost always apical rather than sternal and may be of a dull, constant aching character or momentary and stabbing When there is shortness of breath it may be merely of the type that accompanies a state of fatigue as if it were a greater burden to lift the chest or it is due to a peculiar 'sighing type of breathing This latter phenomenon is sufficiently common and characteristic to require special emphasis We frequently see patients, often young women, who complain of shortness of breath without any other evidence of heart disease On questioning them it will be found that the dyspnea occurs particularly at rest It is obvious that true cardiac dyspnea at rest indicates a very grave condition and yet these individuals appear quite well On further questioning they will describe the sensation as air hunger and say 'I just can t get enough air' During the examination the physician is apt to catch them in the act, so to speak, and see them occasionally take a very deep breath like a sigh At that very instant it is well immediately to ask the patient if that is what is troubling her This enables one to identify this primary complaint as functional for the patient actually overventilates the lungs for some peculiar reason and still does not feel that sufficient air is obtained Even minor changes in the electrocardiogram such as very slight inversion of the T waves in any of the leads and slight depression in the S-T segment have been observed in some cases When this condition is marked and maintained it may result in sufficient overventilation to produce symptoms of tetany The patients may complain of tingling and numbness of the extremities, show carpopedal spasm and a positive Trousseau (spasm of the fingers on squeezing the forearm) or Chvostek sign (contraction of the face on tapping the facial nerve) A proper explanation and assurance that nothing serious is going on often is sufficient to cure the patient

There are other features that are present in these functional cases that deserve attention Sweating, tremulousness and nervousness together with palpitation make one think of the possibility of hyperthyroidism At times the differential diagnosis is not simple and will require basal metabolism studies Although a somewhat increased heart rate is commonly present, if observations are made while the patient is asleep it will be found to be slow At times the entire picture resembles incipient tuberculosis and constant care should be taken to avoid overlooking a tubercular process by having roentgenograms taken of the lungs when any doubt exists

Scherf has reported a group of cases that would ordinarily be classified as cardiac neurosis or neurocirculatory asthenia, in which he regards the cause to be some disturbance in the endocrine system In these cases

there may even be minor changes in the electrocardiograms with flat or slightly depressed S T intervals Scherf believes these patients can be helped or cured by the administration of estrogenic hormone

Neurocirculatory Asthenia

The functional cardiac disease that is often designated 'neurocirculatory asthenia is sufficiently important to merit more detailed discussion This condition, first described during our Civil War, was very prevalent in the First World War It was interesting to observe the different circumstances under which the symptoms developed Some of the soldiers first began to show evidence of an unstable neurocirculatory apparatus when they first appeared before the examination boards, when they were not yet drafted into the army The very thought of becoming a soldier and enduring all the hardships that it entails was sufficient to produce palpitation, weakness, chest pain, tremulousness, sweating, giddiness and dyspnea Others first showed these symptoms while they were training in the camps Still others did well until they were sent overseas At the other extreme, there were men who carried on sturdily for two or three years, going through terrible experiences in the trenches without showing any evidence of neurocirculatory asthenia and then finally the nervous reserve would become exhausted As a last straw it might be a simple event which had never previously disturbed them, like a shell explosion, that precipitated the symptoms It is not surprising that all these different gradations were found during the First World War If this condition was more frequent among the British soldiers than among the American, and from my own observations I believe this was true, it probably was due to the fact that the British had to undergo the terrific strain of warfare longer than the Americans and that in another year or two we would have had to equip special hospitals to look after these functional cardiacs as did the British

Probably the most important factor in predisposing to this disease is the constitutional factor Many have a past history or family history of a subnormal nervous makeup It is common to elicit a story of nervous breakdown, fainting spells, psychosis, neurasthenia, epilepsy and the like in the past or family history of the patient With this background it may then be necessary to have a precipitating cause to bring to the surface the symptoms of functional heart disease The relative importance of those two factors—predisposing and precipitating—will vary in different individuals No doubt if one factor is sufficiently prominent the other need be very slight to produce the symptoms During the war, when the psychic trauma of combat was most intense and prolonged, individuals without any detectable evidence of a constitutional defect in their nervous stability finally developed soldier s heart Others with a marked background of nervous instability would break down at the slightest provocation During the First World War the precipitating causes were linked up primarily with fear and less frequently with infection Gunshot wounds, shell explosions, being buried, gassing, ' trench fever and rheumatic

fever made up the common direct causes that precipitated the symptoms of neurocirculatory asthenia In civil life the same hereditary constitutional factors are present but the direct precipitating causes are anxiety following the loss of one s fortune, the death of some friend or relative, a love affair, or any state conducive to fear or emotional and nervous tension

The symptoms previously enumerated that accompany neurocirculatory asthenia mainly concern the cardiovascular and nervous systems Because for the most part these symptoms are the very ones that are brought on by effort in normal people, the condition has also been called "effort syndrome ' The difference is that in these patients the effort required to produce the symptoms is inordinately slight as compared to that necessary to produce them in normal individuals Easy fatigability, breathlessness, palpitation and precordial pain are the most common complaints Those afflicted cannot stand much physical or mental activity On examination very little of importance is discovered Some have a peculiar expression of fatigue in their faces They may show cyanosis of the face and hands which is due to local sluggishness of the circulation The heart shows nothing abnormal except occasionally a slight systolic murmur Not only is the heart not enlarged but there is a tendency for it to be small as determined by x ray examination In most, the electrocardiograms are normal but occasionally slight flattening or even inversion of the T waves in Lead I or II may be found There may be a tremor of the hands and a moist skin All that has been said concerning neurocirculatory asthenia or soldier s heart, which is so prevalent in soldiers, is applicable to civil practice, only the clinical manifestations are less prominent in civil life

The importance of this condition is that it must be recognized and not confused with organic heart disease So often the physician errs and makes a ' cardiac cripple out of one who is structurally sound A patient with only minor functional complaints may become much worse and even invalided unnecessarily as a result of a mistaken diagnosis The physician, believing that he is dealing with some valvular or myocardial disease, cautions the patient against overactivity or may even direct that he stay in bed This convinces the sufferer that his heart is diseased and from then on he becomes more and more introspective and the symptoms become aggravated It may subsequently become very difficult or impossible for some other physician to convince him that his heart is sound Having made the diagnosis of functional heart disease one should speak with emphasis and assurance It is not sufficient to tell the patient Your heart is normal, but don t overdo ' Or in reply to the question "May I play tennis? if the physician says ' No, I think that is too strenuous any intelligent patient has a right to feel that the physician is holding something back from him or is not certain of his ground One should reply "You can do anything you please, even if you overdo and feel tired no harm will come From this he may become convinced that there is nothing to fear and thereby make the most important step in his recovery

Apart from this assurance it is well to try to disentangle any disturbing psychic factors that may be playing a role All other general hygienic influences conducive to good health should be gone into such as graded exercise and particularly the question of nutrition In those who are underweight a gain of weight proves very helpful

A few words may be appropriate concerning the relation between neurocirculatory asthenia and warfare My own personal experience in World Wars leads me to the opinion that soldiers with this condition added very little to our efforts as actual fighting men Once symptoms were present it is unlikely that any were able to carry on front line duty Some men with milder symptoms who improved under a course of graded exercises were sent back to the trenches Not many were able to carry on More often, after a few days of shelling back they would appear in one hospital after another and occupy bed space for months The obvious inference is that when the diagnosis is made such a man should not be accepted for military service at all or only for limited duty behind the active front This policy would be wise until the need for men is so great that second rate soldiers have to be drawn It must also be appreciated that in making the diagnosis, symptoms are more important than signs, for it is the complaints they have, such as breathlessness syncope, weakness palpitation and precordial pain that incapacitate them

A wrong opinion prevailed amongst most physicians concerning prognosis It was thought the moment war ended and the fear of suffering or death was over, that the symptoms would all disappear Follow up studies showed that not to be the case Although many men improved many others retained their symptoms to a greater or lesser extent indefinitely This could not be explained entirely on the desire these veterans had to receive pensions, for some who did not need or accept pensions continued to be handicapped

PROGNOSIS

Prognosis in cases of functional heart disease is uncertain with regard to the symptoms but excellent as to life Although one does not fear any serious complications and there is no mortality due to this condition cures are not readily obtained Often the underlying fear or psychic disturbance cannot be removed or the symptoms go through cyclic changes improving and then reappearing as different periods of nervous stress and strain come and go Even without any obvious cause some patients will continue to be bothered by one or another of these functional symptoms as evidence of some neurocirculatory instability Because of the great fear of heart disease that prevails among the laity, patients with functional heart disease need constant reassurance and encouragement to carry on

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Paroxysmal Rapid Heart Action

The heart action may suddenly become rapid in a variety of ways. There are several different types of disturbances in the heart mechanism responsible for these paroxysms. It is important to distinguish one from the other because, as we shall see later, the treatment and the prognosis differ considerably in the various types. Furthermore, there are occasional instances in which proper treatment must be instituted quickly, otherwise disastrous results ensue. This whole subject, therefore, has many very practical aspects, and it is surprising how intelligent management of these disturbances can be carried out by the use of very simple means.

Paroxysmal rapid heart action comprises those conditions in which the heart suddenly becomes rapid. The termination of such a paroxysm is likewise sudden. If one had the opportunity of observing the actual transition either at the onset or the offset of these attacks, one would notice that the change takes place instantly. A heart rate which is found to be 70 at one moment might suddenly in one second jump to 200 a minute and at the termination of such a paroxysm the rate would fall in one beat from the high level to a normal one. The abruptness of the change is an important point that distinguishes it from a rapid heart in which the mechanism is normal, i.e., normal tachycardia. If a patient is seen with a normal sinus tachycardia of 170 for instance, as might be found in surgical shock, hemorrhage, certain fevers, hyperthyroidism and other conditions, it generally will be possible to ascertain that the heart rate rose gradually to this high level. It may have taken minutes, hours or days to change from the normal to the rapid rate. In true paroxysmal heart action no such gradual transitions occur.

The cause of these paroxysms is the inception of a new cardiac mechanism. In the ordinary normal rapid heart the rhythm is really not disturbed. The impulses arise in the normal pacemaker (sino-auricular node) and travel through the heart normally. The only change is that the rate of impulse formation is rapid. In these abnormal paroxysms, on the other hand, the ordinary pacemaker of the heart no longer controls the rhythm and in its place some abnormal mechanism in the auricles or ventricles controls the heart beat. The types of disturbance that may result are

paroxysms of auricular tachycardia, auricular flutter, auricular fibrillation and ventricular tachycardia Although paroxysms may also arise in the auriculoventricular node or the bundle of His and are then called nodal tachycardia, they are rare, and are impossible to recognize without the use of special apparatus

The following conditions are also considered from an electrocardiographic point of view in Chapter 21

PAROXYSMAL AURICULAR TACHYCARDIA

Paroxysmal auricular tachycardia is the most common of these disorders It occurs much more often in patient who have no heart disease than in those with organic disease In this sense it may be regarded as a functional disturbance The heart rate during an attack will generally range between 150 and 250 a minute The attack begins and ends abruptly It may last minutes, hours, days or rarely weeks During the paroxysms the heart rate is perfectly regular and the sounds are all alike in quality and intensity The regularity is so precise that on careful measurement contiguous heart cycles will not differ in length by more than $\frac{1}{100}$ second The rhythm is not only regular but seems to be constant and fixed at the same level for long periods at a time If an attack were to continue for some hours the heart rate might be found to be 196 at one time and ten minutes later it would still be 196 It is also impossible to disturb this rate by such simple means as having the patient hold his breath or change his position, which measures generally will alter the rate of a normally beating heart This constancy of rate is an important distinguishing characteristic which enables one to recognize the condition at the bedside To obtain this the apex rate should be counted accurately for sixty seconds This can be done with a stethoscope in human beings, when the heart is regular, even when the rate is as rapid as 250 a minute The error in the count need not be greater than two one at the beginning and one at the end of the minute while trying to synchronize the heart beat with the second hand of the watch During the intervening beats there need be no inaccuracy whatever Accurate counting of a rapid regular heart may be facilitated by simultaneously tapping with the foot or the finger This matter deserves some emphasis because one often hears the expression that the heart was too rapid to count If the rhythm was regular this generally means that the observer made no real attempt to count accurately Let us assume that in a given case the rate of 164 was found Some minutes later the count should be repeated having the patient change his position, hold his breath or go through any maneuver which ordinarily disturbs the heart rate On the recount the rate will again be found to be 164 or 163 If the second rate were 170 or 160 one might fairly safely assume that the patient did not have paroxysmal auricular tachycardia A normally accelerated heart, however, might alter its rate to that extent Thus we have a simple means which helps to characterize this condition Expressed in other terms, while auscultng the heart not even the slightest

alterations in the rhythm will be noted as a result of those measures which generally produce acceleration or retardation under normal circumstances

Symptoms

The clinical features in this condition are variable The attacks may occur rarely, one every several years, or frequently I have seen instances in which the patient had as many as ten paroxysms every day He will generally complain of sudden palpitation of the heart and become nervous and agitated Fainting and actual loss of consciousness may occur at the onset of any form of paroxysmal rapid heart action This probably results from the sudden decrease in the cardiac output with resulting relative cerebral anoxemia Adjustments in the circulation take place, however, so that syncope does not last very long During the first time or two, not knowing what it all means, he may actually fear that he is going to die Usually there is no dyspnea with the paroxysm and after a variable length of time the attack ends as suddenly as it commenced There frequently is a sensation of gaseous distention of the abdomen or belching of gas during and particularly at the end of the paroxysm Occasionally a cessation of the attack comes with a vomiting spell These features make the patient think that the paroxysm is due to indigestion' or that it is in some way related to his diet The causes that precipitate the attack are numerous and inconstant Frequently a sudden movement of the body such as bending to tie one s shoe or a quick turn of the head initiates the attack Much less frequently violent effort brings on a spell Emotional factors also come into play Sudden thoughts or even dreams may be the precipitating cause There remains a great number of instances in which the attack seems to occur spontaneously without any known cause In some cases there is heart pain during a paroxysm which is more apt to occur after the paroxysm has lasted some time In its description it may resemble the pain of angina pectoris but it has by no means the same prognostic significance, for when the heart rhythm is normal and slow there will be no chest pain whatever and in fact no other evidence of heart disease

Physical examination of the patient during an attack is apt to reveal no essential abnormality except the rapid heart rate The patient may seem pale and agitated and the skin may be moist When an attack develops in one who is otherwise normal there will be no evidence of circulatory insufficiency except in very rare instances This is also true in some cases in which there is associated organic heart disease, either valvular or muscular In fact, there is no other condition in which the heart may be so rapid with so little apparent embarrassment to the circulation The patient may have no dyspnea or cyanosis and yet show a heart rate of 200 or more

Under certain circumstances symptoms of considerable gravity develop, such as peripheral thromboses and congestive heart failure The factors that determine the development of these complications are the following the duration of the attack, the heart rate during the attack and the condition of the heart before the onset of the attack It is obvious that a normal

heart might stand the rate of 240 a great deal longer than a heart with mitral stenosis, or one with a poor degree of compensation I have seen a patient with mitral stenosis develop marked dyspnea, edema of the lungs, cyanosis and engorgement of the liver within a few hours after the onset of tachycardia in which the heart rate was 190 On the other hand, if the rate is extremely rapid and the attack of long duration, even if the heart is structurally normal disastrous results may develop This was well illustrated in a case I observed many years ago A man of 40 who was otherwise perfectly well had had three attacks of tachycardia during the previous few years Each attack lasted uninterruptedly for five to eleven days During the first one he developed a hemiplegia from which he gradually recovered in the course of a few months with a slight residual spasticity of one side of the body During the second attack he developed aphasia which gradually disappeared in four months During the third *attack, dry gangrene of the left arm developed requiring amputation at the* shoulder I saw this man in 1914 during the fourth attack and found the heart rate to be 250 a minute After obtaining certain data on this patient the attack was immediately ended by ocular pressure It was the first time his attacks had been controlled for the others had stopped spontaneously after lasting many days In fact this may have been the first instance in which ocular pressure was ever effective as a treatment for paroxysmal tachycardia

The explanation of the complications that occurred in this case was quite obvious While the heart was beating 250 times a minute the pulse pressure was extremely low During several attacks in which the patient was observed the systolic pressure was around 94 to 96 and the diastolic around 88 mm Therefore, this patient had an effective pulse pressure of no more than 6 or 8 mm One can readily see from this how thrombosis in peripheral vessels could easily develop This must have occurred in the cerebral vessels during two of the attacks and in the vessels of the left arm at the time gangrene occurred The process essentially consisted of stagnation of the blood In such a case it is evident that proper therapy was imperative, for this patient lived for more than thirty years thereafter

In general, the changes in blood pressure that occur during paroxysmal tachycardia consist in a tendency for the systolic level to fall and the diastolic to rise with a diminution in the pulse pressure The degree of such changes will depend upon the three factors mentioned, namely, the heart rate the duration of the attack and the structural condition of the heart Such attacks have at times been called acute dilatation of the heart This is a misnomer for in a series of twelve such instances x ray measurements of the heart before, during and after attacks failed to show an appreciable dilatation except in one instance (case cited in the preceding paragraphs in which the heart rate was 250) In some patients the heart was actually smaller during the attack Other interesting findings that may develop during attacks of tachycardia are a slight fever and a slight leuko cytosis I do not think that these features indicate infection They are rather the result of congestion

One can readily see how a simple attack of any type of paroxysmal rapid heart action may be confused with acute coronary thrombosis A patient has a 'heart attack The heart rate is found to be very rapid There may be some collapse, a fall in blood pressure and even chest distress of the anginal type Fever and leukocytosis may follow After the attack has ended the electrocardiograms may even show inversion of the T waves, suggesting coronary thrombosis These changes are probably the result of myocardial fatigue and local relative anoxia and gradually return to normal in subsequent days or weeks All this can occur in a structurally normal heart and also leave the heart entirely intact The confusion becomes even greater when one considers the possibility that actual coronary thrombosis may result in vulnerable individuals if a low blood pressure and a state of shock persists for a long time One important distinction is that in simple paroxysms of tachycardia the very rapid rate comes first and then the above signs and symptoms follow, while in coronary thrombosis the abnormal tachycardia follows the attack and generally does not appear for one to several days

Treatment

The development of paroxysmal tachycardia may present a serious situation and require immediate and effective treatment during labor or during a surgical operation I recall an experience in which a woman was being operated on for gallstones While she was under ether, just as the operation was to be started, respiration ceased, the radial pulse became imperceptible and extreme cyanosis quickly developed Artificial respiration had to be instituted and had already been carried on for about ten minutes when I saw the patient The heart rate was 212 and perfectly regular Carotid sinus pressure stopped the attack immediately, the heart returning to a normal rate of 80 Normal breathing was immediately restored and the cyanosis disappeared The patient was subsequently operated on and the gallbladder containing stones removed In two other cases that were somewhat similar but in which the symptoms were not so alarming, carotid sinus pressure was not effective whereas ocular pressure promptly restored the heart to its normal rate Such experiences are not common The urgency and gravity of the whole situation is so great when they do occur that they deserve this emphasis

There are two phases to the treatment of paroxysmal tachycardia, the first concerns the treatment of the attack and the second the procedures that may prevent or diminish the incidence of future attacks There are a great many measures which have seemed to be successful at one time or another in stopping attacks They all produce their beneficial effects by stimulating the vagus nerve in one way or another The act of vomiting is one of these, either produced spontaneously or by the use of an emetic, such as apomorphine, ipecac, and the like One to 4 teaspoonfuls of syrup of ipecac *often is effective when other methods fail* and may be repeated in one-half to one hour Inducing the gag reflex by inserting a finger in back of the tongue is another method I knew one patient who stopped

several of his attacks by drinking ice-cold water or swallowing bits of ice Occasionally lowering the head or lifting the foot of the bed has proved successful One simple and most helpful measure is holding a deep breath There are many patients who have found that this simple experiment can stop their attacks very effectively It is well to tell such patients to take a deep breath and hold it as long as they can In normal individuals, holding a forced inspiration produces a slight vagal slowing of the heart but in this condition it will frequently bring an attack abruptly to an end with restoration of a normal slow rhythm (Valsalva experiment)

Finally, there are three methods which stimulate the vagus nerve more effectively The first of these is by irritation of the carotid sinus, the second by pressure over the eyeball and the third by the use of drugs The first of these procedures consists of palpating for the bulge in the common carotid artery as it divides into external and internal branches with one hand while supporting the patient with the other hand placed behind the neck Pressure is then exerted backward, completely com pressing the artery and massaging it for several seconds This should be done first on one side of the neck then on the other, but not on both sides at the same time Pressure should be released after a few seconds and repeated after an interval if necessary In rare cases hemiplegia has resulted which was not due to an asystole of the heart but rather to ob-literation of the circulation to one side of the brain for too long a time The cerebral accident in these cases always involved the side compressed If the paralysis had been due to a cessation of the heart beat one side *or* the other would have been affected The second method consists in exert-ing rather firm pressure with the thumb over the eyeball This will be painful Here again one eye at a time should be tried This pressure sends a stimulus up the fifth cranial nerve and by a reflex (oculocardiac reflex) down the vagus nerve When ocular pressure is effective it is not a result of the pain for pain produced elsewhere in the body causes no such slowing and I have several times stopped attacks while a patient was under anesthesia Its use is limited to some extent but there are instances when this will prove successful after other methods have failed

The distressing feature of arresting attacks of paroxysmal heart action is that on rare occasions the attack stops but the heart never starts beating This experience makes one think that the abnormal rhythm is inhibited but the normal pacemaker, which has not been functioning throughout the attack, fails to initiate any impulses It is as if the sinus node which has been asleep, fails to wake up I have been told about two instances of sudden death, one following carotid sinus pressure and the other after ocular pressure in which the tachycardia stopped but the heart beat never was resumed

Attacks of auricular tachycardia can generally be arrested by one or another of the procedures just described There are instances, however, when the tachycardia is resistant to all these measures Drugs may then be used which have proved valuable in stubborn cases On several occa sions in which the tachycardia persisted for a long time and could not

be stopped by the ordinary means and the patient s condition seemed rather critical, the intravenous injection of quinidine sulfate immediately restored the normal rhythm For this purpose one would generally need 0 3 gm or more of quinidine sulfate It is best to inject this slowly while someone is listening to the heart over the precordium Just as soon as the break in the rapidity occurs, injection should be stopped In two cases I found that after 0 2 gm of the drug was injected, and while the hypodermic needle was still in the vein, the attack suddenly came to an end There is some danger in the use of quinidine, therefore this treatment should not be used until other measures fail and the patient's condition is critical enough to warrant it Another drug that has been used with great success is acetyl-β methylcholine The average adult dose is 20 mg given subcutaneously Infants have responded favorably to doses of 5 mg This is a powerful vagal stimulant Occasionally untoward results occur, particularly in allergic individuals, for which $\frac{1}{80}$ to $\frac{1}{30}$ grain of atropine should be used Other drugs have been used successfully to stop attacks Among these are calcium gluconate (10 to 20 cc of 10 per cent solution given slowly intravenously), parathormone subcutaneously, magnesium sulfate (15 cc of 20 per cent solution intravenously) and prostigmine methylsulfate (0 5 to 1 0 mg intramuscularly) There is reason to believe, however, that calcium might be dangerous in ventricular tachycardia Neo synephrine intravenously has stopped attacks of paroxysmal supraventricular tachycardia within one minute The mechanism probably depends on increasing the blood pressure It might be hazardous in cases with hypertension or coronary disease The dose is 0 5 to 1 0 mg Curiously enough, I observed one patient who had typical angina pectoris and in addition classical attacks of paroxysmal auricular tachycardia The two conditions were independent of each other This patient found that nitroglycerin not only relieved the attack of anginal pain (when the heart rate was normal) but stopped his attacks of tachycardia Finally large doses of digitalis (0 5 to 1 0 gm) often prove successful Probably any form of digitalis preparation will do, though excellent results have been reported by the intravenous use of cedilanid (1 6 mg) Atebrin also has been effective in arresting various forms of tachycardia This is given in doses of 0 1 gm intravenously (1 per cent solution) or 0 3 gm in 1 per cent novocain solution intramuscularly

It must be understood that in most cases of paroxysmal tachycardia the attacks eventually cease spontaneously and that the patient is none the worse for the spell It is only occasionally that any heroic treatment would be indicated, but there are instances in which it might prove lifesaving

When it comes to preventing the return of such attacks, the problem becomes more difficult The patients themselves often feel that if treatment were directed at the gastrointestinal tract they might be cured The physician frequently alters the diet in one way or another, advises medical treatment for gas, indigestion or constipation, with the hope of preventing these attacks In other instances courses of sedatives have been prescribed to diminish the general nervous irritability In those cases in which there

is excessive use of tobacco this may be ascribed as the cause of the attacks Treatment along these lines, however, has always proved practically useless It recently has been suggested that carbachol (carbamylcholine chloride) 4 mg orally daily may prevent recurrences of supraventricular tachycardia I have had no experience with this drug The two drugs that are helpful in preventing recurrent tachycardia are quinidine and digitalis Either one or the other, if properly administered, will often either inhibit the attacks entirely or will diminish their frequency and severity In several instances I have found digitalis to be effective after quinidine had failed In only a portion of the cases is this treatment indicated If attacks come only on rare occasions, such as once in six to twelve months, it would hardly seem wise to prescribe a course of preventive treatment Not knowing when an attack is to be expected, such a patient would have to be constantly under the influence of quinidine or digitalis in order that the heart might be prepared on that distant day when the attack otherwise would be due This would necessitate the constant use of a medicine for the possible prevention of a rare attack Even if months elapsed and the patient were free from attacks because of the infrequency of such spells, it would still remain doubtful whether the therapy had anything to do with the result obtained Under these circumstances it is best to explain the entire situation to the patient assuring him that nothing serious will develop to institute no constant drug therapy, but to be prepared to meet each spell as it arises

When the attacks occur frequently, treatment should be specifically directed at their prevention, for then they may be actually incapacitating and it becomes a simple matter to decide whether or not therapy is effective Quinidine sulfate may be given in doses of 3 to 5 grains three times a day If the patient ordinarily has attacks every few days a particular dose should be continued long enough to be certain whether it is effective or not If attacks continue despite this dose it may be increased Although some patients may tolerate larger doses without showing toxic symptoms, such as ringing in the ears, as a practical matter if attacks continue while 5 grains three times a day are taken it is best to omit the drug entirely

It was formerly thought that digitalis was of no use in the treatment of paroxysmal tachycardia This opinion was held mainly for two reasons In the first place it seemed true that the ordinary administration of digitalis during an attack had little influence in stopping it In the second place its use in preventing the recurrences seemed disappointing because complete and constant digitalization was generally not carried out Some years ago I had an opportunity of observing four patients who had had courses of quinidine and digitalis therapy without success The digitalis in these cases was inadequate and when the dosage was administered in quantities similar to those used in heart failure, all attacks ceased One of these patients was having several attacks a day so that he had to quit his work He had no organic heart disease and except for these attacks he was well Under full doses of digitalis all attacks ceased and he was able to return to work Another patient was a woman aged 60 who had had attacks

about every two weeks for the previous three years They grew increasingly severe so that finally they were absolutely prostrating When she was seen during one of these spells she was found to be pulseless, no blood pressure reading could be obtained and the heart rate was 240 She was cold and it looked as though she might die Ocular pressure instantly brought the heart rate to normal She had previously taken quinidine at one time, and at another 5 drops of digitalis three times a day for some months without success She had also taken sedatives and various medicines for indigestion After the spell just described she was given 1½ grains of digitalis leaves three times a day for one week and thereafter 1½ grains daily There were no attacks for fifteen months It was then decided to stop digitalis to see whether or not it was needed any longer About one week after the drug was omitted a similar attack occurred and digitalis was then reinstituted and the patient had no further attacks This case was a very striking one for the patient remained perfectly well for over fifteen years, whereas formerly for three years she was in constant fear of these attacks, and had to live a very confined life and required constant nursing attention

In the more common type of paroxysmal auricular tachycardia just described, there is no heart block, the ventricles responding to every auricular impulse There is a much rarer variety in which block does occur In this and other ways this type resembles auricular flutter to some extent It will not respond very readily to the methods of treatment just mentioned Quinidine or digitalis may be tried, however, for occasionally success may be obtained It is desirable at least to keep the ventricular rate slow by constant use of digitalis, if possible, for the rapid auricular rate may persist for weeks or months New observations by Wilson and his associates have presented evidence suggesting that this type of auricular tachycardia and possibly others as well are due to a circus movement in the auricles, in which the pathway goes through the auriculoventricular node

Surgical procedures have recently been employed in stubborn cases of recurrent paroxysmal auricular tachycardia and in other types of paroxysmal rapid heart action Either the right or the left or both sympathetic chains from the lower cervical through the upper four or five dorsal ganglia have been removed It is too early to be certain of the results but they seem promising, although it is already evident that failures will occur

PAROXYSMAL AURICULAR FLUTTER

Another form of paroxysmal heart action is auricular flutter Although this condition once established is apt to be persistent, occasionally it occurs in paroxysmal form Here the auricular rate generally ranges from 250 to 350 and the rate of the ventricle is one half of the auricular rate or less In very rare instances the ventricular and auricular rates are the same When the condition is first seen, before any treatment is instituted, there is apt to be a 2 1 heart block, so that whatever the auricular rate may be, the heart rate as counted at the apex is *half as great* The former

may be 340 per minute whereas the ventricular rate is 170 It should be borne in mind that the rate of the auricle is really inferred, for the auricular contraction cannot be heard The rhythm in untreated patients is generally regular, because every second impulse coming from a regularly beating auricle reaches the ventricle Occasionally in untreated cases and frequently after treatment has been started the rhythm is irregular Here the degree of block is changing in different heart cycles, so that for a short time every second auricular beat gets through to the ventricles, then only the third or fourth beat gets through and so the regularity is disturbed from time to time The resultant irregularity may occasionally be gross enough to resemble auricular fibrillation very closely There may even be an appreciable pulse deficit The essential difference between the two conditions is that in auricular fibrillation the irregularity is actually complete whereas in flutter it follows some definite law, i e, different groups of ventricular beats will have the same length if accurately measured and properly selected as they will correspond to the same number of regularly occurring auricular cycles (Chap 21, Fig 68)

This type of paroxysmal rapid heart action occurs generally in patients who have organic heart disease, either valvular or myocardial, but may also occur in those with no other evidence of heart disease Unlike paroxysmal auricular tachycardia it is apt to persist for long periods at a time or even to remain permanent if untreated Therefore it is important to try to restore the normal mechanism or at least to control the ventricular rate There are two purposes in the treatment of patients with this condition It would be desirable to do away with the mechanism of auricular flutter entirely or to prevent the ventricles from becoming rapid if the flutter persists In general the two drugs that are used for these purposes are digitalis and quinidine In about one third of the cases the heart is restored to normal rhythm if digitalis is properly given At first, as has been mentioned the ventricular rate is apt to be rapid and regular with a coexisting 2 1 heart block After the customary dose of digitalis is administered the ventricular rate slows and may become irregular as a result of an increase in heart block so that a 4 1 or 6 1 auriculoventricular block results If the ratio between ventricular and auricular beats is constantly 4 1 or 6 1, the rhythm is regular, if this ratio is changing in different cycles the rhythm is irregular The ventricular rate may now be 60 or 80 but the auricular rate during this time remains unchanged, as the flutter so far is not disturbed At this point in some cases, auricular fibrillation may suddenly develop with the appearance of a gross irregularity If digitalis is now omitted, during the course of the next day or so the heart may return to a normal rhythm In fact, regularization may take place even if the maintenance dose of digitalis is continued In general, therefore digitalis may change the auricular flutter into auricular fibrillation and permit the auricles to return spontaneously to a normal rhythm When this treatment is ineffective, auricular flutter may persist even though the ventricular rate has been slowed by digitalis, or having produced a change to auricular fibrillation this may continue indefinitely, or again

return to the original state of flutter When digitalis does not restore the normal rhythm it may still serve an important therapeutic purpose in maintaining a slow ventricular rate, whether the original flutter or the subsequent fibrillation persists Inasmuch as the most important purpose of treatment is the prevention of a rapid ventricular rate, no matter what the auricles are doing, digitalis is of distinct value in this condition

The best treatment of auricular flutter is actually to restore a normal rhythm It is known that quinidine increases the refractory period of auricular muscle By so doing it may actually stop the continuous circus which exists in the auricle and which tends to perpetuate the flutter The dose of quinidine for this purpose is not fixed In general, from 5 to 10 grains three to four times a day may be given The procedure I have used is, after digitalis has slowed the ventricular rate and the abnormal rhythm persists, to start with a dose of 0 2 gm and to increase it by 0 1 gm with each dose, giving the medication three times a day During the quinidine therapy a daily dose of 0 1 gm of digitalis may also be given At times I have had to increase the single dose of quinidine to 1 0 or even to 1 5 gm before reversion to normal rhythm was obtained It is desirable to have the patient under close observation during this time so that frequent electrocardiograms may be taken It may be noted that during the quinidine administration the actual auricular rate will slow while the ventricular rate accelerates I have seen the auricular rate of flutter drop from about 300 to 150 as a result of quinidine At any time during this treatment the normal mechanism may suddenly be restored The drug is by no means effective in all cases, and unlike digitalis has no value in slowing the ventricular rate except when it restores the normal rhythm The disadvantages of quinidine apart from its frequent ineffectiveness are that it has certain toxic actions that occasionally are serious On the other hand there are instances of auricular flutter that are helped greatly by this drug when digitalis has been ineffective

PAROXYSMAL AURICULAR FIBRILLATION

Paroxysmal auricular fibrillation is a much more common condition than it was thought to be not so long ago In fact the term auricular fibrillation is applied to a condition that was formerly called perpetual arrhythmia" It was then believed that once this became established it remained so permanently We now know that the transient form is quite common It occurs occasionally in patients with mitral stenosis before it takes on the permanent form It is also frequently met with in hypertensive heart disease or in so-called 'chronic myocarditis It occurs in some cases of rheumatic fever and in a small proportion of cases of pneumonia It has been frequently observed during acute coronary thrombosis Rarely it is seen under a variety of circumstances that seem to have no very definite relations to the heart, e g , acute angioneurotic edema, chronic gallbladder disease or as a complication of any surgical operation requiring general anesthesia, especially in operations on the lungs and mediastinum Possibly the most common condition with which transient

auricular fibrillation is associated is hyperthyroidism This is a frequent event during the ordinary course of the disease and particularly during the first day or two following an operation on the thyroid gland There remains a small group of patients in whom transient fibrillation occurs and in whom there can be detected no other evidence of heart disease or disease of any important organ It must be regarded in these isolated cases as a purely functional derangement and not indicative of any disease It is evident from the great variety of conditions in which transient fibrillation may occur that the symptoms resulting from this disorder and the appropriate treatment must vary a great deal It is obvious that the sudden inception of a rapid irregular rate can produce very distressing symptoms in a patient who already has serious organic heart disease, whereas if it occurs in a heart that is essentially sound very little embarrassment may result As will be seen later, in some cases this paroxysmal arrhythmia needs no treatment whatever or treatment may be entirely useless until the underlying cause is removed, whereas in other instances the attack itself presents a therapeutic problem of major importance

When auricular fibrillation develops the rhythm of the heart becomes grossly irregular Almost invariably the rate as counted at the apex is quite rapid, 130 to 170 or more If the patient is digitalized or if, as rarely happens, there is a concomitant defect in the conduction apparatus the apex rate may be slow With a rapid heart rate there generally is an appreciable pulse deficit, i e, the pulse rate is 10 or more less than the heart rate Occasionally when there is hypertension or when the pulse pressure is great, as in hyperthyroidism, or when the heart rate is not very rapid there may be very little if any pulse deficit The bedside diagnosis, however is not very difficult in the great majority of cases The most important feature in the diagnosis is the character of the irregularity It is a complete absolute and tumultuous arrhythmia that one hears Only in rare instances may it be confused with other conditions

The symptoms that result from an attack of auricular fibrillation depend in a great measure on the state of the circulation before the onset of the attack In most cases there is palpitation This is produced by the rapid agitated contractions of the heart If cardiovascular disease already exists, dyspnea of a mild or marked degree may quickly develop In fact some cases may present the picture of acute pulmonary edema Occasionally an attack of transient auricular fibrillation is associated with the dislodgment of an embolus from an auricular thrombus If the thrombus is dislodged from the right auricle a pulmonary infarct develops and if from the left auricle hemiplegia or other arterial infarction may occur If auricular fibrillation develops in a patient already very sick with pneumonia sudden collapse may occur I recall one such case in which the heart rate suddenly rose to about 200 and the pulse became imperceptible The patient seemed to be *in extremis* Twenty minutes after an intravenous dose of strophanthin he quickly revived, the heart rate fell to about 110, the pulse returned and the patient eventually recovered There are other instances of transient fibrillation in which the circulation is in such good condition

that the patient will have no complaints whatever, except a slight degree of palpitation One can readily see how varied the picture may be

Treatment

There are two therapeutic aspects to this condition, one concerns itself with the underlying disorder, whether it be pneumonia, exophthalmic goiter or some other condition, and the other is the treatment of the specific attack of fibrillation In hyperthyroidism it is useless to treat the patient for auricular fibrillation in the ordinary way, as attacks will tend to recur as long as the hyperthyroid state persists When the treatment of hyperthyroidism is effective in restoring the metabolism to a normal level the tendency to fibrillation will generally disappear spontaneously In most other types of transient fibrillation direct therapy for the arrhythmia itself is indicated The two drugs that have distinct therapeutic value in the treatment of this arrhythmia are digitalis and quinidine The action and the purpose of these two drugs are quite different and so are the results obtained from their use Digitalis rarely brings an attack of auricular fibrillation to an end When such a spell ceases while the patient is taking digitalis it is thought to do so spontaneously In fact, there is reason to believe that digitalis might tend to perpetuate the condition The value of digitalis, however, is to slow the ventricular rate while the fibrillation continues On the other hand, quinidine may even cause an acceleration of the ventricular rate while fibrillation is present, but its main value lies in the fact that it tends to do away with the arrhythmia entirely and restore the heart to a normal mechanism If it is known that the patient has had previous spells of this arrhythmia of short duration lasting only a few hours, and the general state of the circulation is quite satisfactory, it is just as well to give neither one nor the other form of medication, but rather to administer some sedative or possibly morphine, for in a few hours the attack may be over If, on the other hand, the patient already has sufficient organic cardiac disease so that the rapid irregular ventricular rate has already produced or may readily produce generalized congestion or alarming symptoms of circulatory insufficiency, rapid digitalization is indicated The reason for this is that it cannot be known how long the attack is going to last or whether or not the fibrillation will become permanent Whether digitalis shall be given by mouth, intramuscularly or intravenously depends upon the urgency of the situation The following is an illustration of this problem A middle aged woman with fairly well-compensated mitral stenosis had, during the course of several years, a few transient attacks of auricular fibrillation, each lasting about an hour or two During these years dyspnea had been slowly increasing Finally she had a spell which had already lasted about five hours when I saw her In this brief time her condition had become alarming She was semistuporous, had marked acute pulmonary edema, striking generalized cyanosis and extreme dyspnea The heart rate was about 190 and the pulse was practically imperceptible The situation seemed desperate Inasmuch as she had not been taking digitalis, 8 cc of digifolin (0 8 gm

digitalis) was immediately given intravenously In about twenty minutes, although the auricular fibrillation still continued, the heart rate fell from 190 to about 100 The change in the appearance of the patient was most dramatic The dyspnea, cyanosis and pulmonary edema all quickly improved and she fell back into a comfortable sleep As it happened, fibrillation from then on persisted She continued on digitalis by mouth and later became ambulatory To be sure, in most cases no such alarming symptoms result In general, palpitation is the prominent complaint and under such circumstances digitalization by mouth is sufficient In a case of moderate severity 0 5 gm of digitalis may be given by mouth in one dose and if none has been previously given, this same dose may be repeated in several hours and again the following day if the heart rate is still rapid Such a procedure will be effective in relieving the symptoms of circulatory insufficiency It is evident from the foregoing discussion that digitalis should be used to slow the ventricular rate when that rate is rapid, when the attack seems to be persisting and when embarrassment to the circulation results

The second phase of the treatment of paroxysmal auricular fibrillation is the prevention or the diminution in the frequency of further attacks In most cases it is best to try the effect of quinidine sulfate This may be given in doses of 0 3 gm three times a day It should be continued for a sufficient time to ascertain whether attacks are being inhibited or not If they had been coming once or twice a week and if while the patient was taking quinidine none had occurred for several weeks, it is fair to say that the attacks were specifically inhibited by quinidine The drug might then be continued for several months If, however, the attacks persisted with equal frequency on this dose, it generally means that quinidine was of no value, although a larger dose might be tried Occasionally attacks have a tendency to recur at certain times of the day or night In these cases the dose of quinidine may be given an hour or so before the expected attack and prove helpful If quinidine sulfate is ineffective as a preventive and the attacks are troublesome or frequent the patient should be digitalized thoroughly and kept on maintenance doses constantly This need not prevent the return of the spells, but will so prepare the heart that when they do occur the ventricular rate will not be rapid In one such case when the attack occurred before digitalis was given the heart rate was about 140 A few days later after only 1 gm of digitalis had been taken by mouth a similar attack occurred and the heart rate was about 100 The following week when the patient was completely digitalized, on routine examination the rhythm, which previously was regular was found to be grossly irregular with a rate of 70 The patient was entirely unaware that she was having a spell, whereas previously spells caused considerable palpitation This experience clearly illustrates the effect of digitalis, for the spells continued but the resultant symptoms were obviated It is also a good example of the slowing effect of digitalis on the ventricular rate in the absence of heart failure, for there was no evidence of congestion in this case There will be occasional instances in which digitalis may be used in the treat-

ment of one specific attack and later quinidine could be given to prevent occurrences It must be borne in mind that many spells pass off without therapy

PAROXYSMAL VENTRICULAR TACHYCARDIA

The last type of paroxysmal rapid heart action to be considered is paroxysmal ventricular tachycardia In this condition the beats arise in the ventricle itself and the auricles either contract at a different rate or in some instances there is reversed conduction so that the impulse that arises in the ventricle travels upward to make the auricles contract There are several important differences between this type of tachycardia and those arising in the auricles This condition in the great majority of cases is associated with serious structural heart disease, generally coronary thrombosis There are occasional instances of its occurring with chronic valvular heart disease and rare cases in which the heart is structurally normal In fact, instances have been reported in which this arrhythmia would develop in the upright position and disappear on lying down (orthostatic paroxysmal ventricular tachycardia) It may also be precipitated by excessive digitalis therapy, but this is more likely to occur where there is already considerable structural damage to the heart Unlike auricular tachycardia, which is often unassociated with any serious heart disease, it may generally be assumed that ventricular tachycardia indicates a serious heart disorder

It formerly was thought that one could not possibly distinguish at the bedside the ventricular from the auricular type of tachycardia It was considered necessary to have electrocardiograms to identify them During recent years, however, some simple clinical criteria have been discovered which enable one to make fairly accurate diagnoses using only those means that every physician can readily employ in the sickroom It has been emphasized that in auricular tachycardia the rhythm is perfectly regular There are no interruptions in the rhythm In fact the differences in length of contiguous heart cycles, if measured accurately, would not be more than one-hundredth of a second In ventricular tachycardia, although the rhythm may seem to be absolutely regular for stretches of many seconds or even of minutes, one may detect, on careful auscultation, in many cases, occasional irregularities Secondly, in auricular tachycardia the heart sounds of the consecutive regular rapid beats are all alike, whereas in ventricular tachycardia every now and then the first heart sound will vary in intensity or quality It will become accentuated or muffled or reduplicated This is due to the inconstant relation between the time of ventricular and auricular systole Another distinguishing feature is the jugular pulse Here one may see fewer auricular beats than the ventricular rate as counted over the precordium, and the jugular waves are often prominent because the auricles may be contracting while the ventricles are in systole Finally, the various measures employed to stimulate the vagus nerve which may end an attack of auricular tachycardia will never influence the rhythm of ventricular tachycardia These criteria together

with the general clinical differences such as the association of the one with functional heart disease and of the other with grave coronary disease should make it possible to recognize ventricular tachycardia fairly accurately even without special apparatus

The beginning and ending of an attack of ventricular tachycardia are abrupt and instantaneous just as in other forms of abnormal rapid heart action The rate during the attack is usually between 150 and 200 but occasionally a higher rate is reached When it occurs without organic heart disease these individuals may complain merely of palpitation although even this may be incapacitating When, as more frequently happens, it develops during the days that follow an attack of acute coronary thrombosis it presents a serious complication of an already grave condition This complication may of itself prove fatal Proper recognition of the condition becomes very important inasmuch as treatment may be effective in restoring the heart to a normal rhythm

Treatment

It has just been mentioned that measures like carotid sinus pressure or ocular pressure are ineffective in arresting ventricular tachycardia Likewise digitalis has no beneficial effect on this condition In fact there is evidence that digitalization may tend to prolong such attacks and accelerate the rate further I studied one instance in which the heart rate rose from 145 to 180 as the patient was given full doses of digitalis The drugs that can restore a normal mechanism when ventricular tachycardia is present are quinidine and pronestyl There is reason to believe that the mechanism involved in the condition is a circus movement just as it is in auricular fibrillation and auricular flutter, only in the former the circus is in the ventricles and in the latter it is in the auricles It is logical to infer that the action of quinidine in breaking up this circus is likewise the same in those two conditions It has been my experience that quinidine has been effective in a larger proportion of cases of ventricular tachycardia than in those of auricular fibrillation

The therapeutic dose of quinidine in this condition is variable and has to be determined individually in different cases One patient who had no organic heart disease but complained of frequent attacks of palpitation, which were proved to be due to ventricular tachycardia, remained entirely free from attacks as long as she took 0 3 gm of quinidine three times a day Other instances, in which the attacks occurred during coronary thrombosis, were controlled by doses of 0 4 and 0 6 gm three times a day One patient with mitral stenosis auricular fibrillation and ventricular tachycardia required 0 8 gm to stop the tachycardia and doses under 0 8 gm three times a day failed to prevent a return of the condition He maintained a normal rhythm for many months while taking the larger dose (0 8 gm) Finally, one very striking case of coronary thrombosis in which this treatment was actually life saving required 1 5 gm five times a day for several days both to stop an attack and to prevent its return, smaller doses having proved to be ineffective In the last instance we dared

to give such large doses because the condition of the patient seemed otherwise hopeless and it was found that smaller doses had had a partial effect in slowing the ventricular rate from 200 to about 150 This slowing of the ventricular rate on increasing doses of quinidine is almost a constant finding At times the rate keeps slowing with each large dose, only to return to the original level as the effect of the drug wears off On rare occasions a large dose of atropine given subcutaneously while the rate was partially slowed by quinidine promptly restored the heart to a normal rhythm In selected cases quinidine may be given intramuscularly or even intravenously Quinidine hydrochloride (Brewer) and quinidine lactate (Lilly) may be used for this purpose It has been suggested that atabrine given intramuscularly in doses of 0 3 to 0 6 gm in 10 cc of 1 per cent novocain has caused regularization in cases of auricular fibrillation, flutter and in ventricular tachycardia in one or two hours when quinidine has failed The physician will have to weigh the circumstances carefully before employing methods of therapy that entail some risk It is, therefore, evident that the judicious use of quinidine in ventricular tachycardia may be of considerable value and on rare occasions life-saving

Magnesium sulfate (2 0 to 4 0 gm intravenously) may also stop ventricular tachycardia instantly I have seen patients in whom the attack stopped within one minute In others it failed when quinidine administered intravenously was successful Another treatment suggested is the intravenous use of morphine The dose given is about 0 015 gm and may be repeated in one-half to two hours The effect is supposed to take place in thirty to sixty minutes As yet I have had little experience with this treatment Potassium salts on very rare occasions may be helpful I saw a patient who had ventricular tachycardia for six weeks He had failed to respond to large doses of quinidine given orally and intravenously Finally reversion to a normal rhythm occurred after three doses of potassium acetate (2 0 gm each) given orally at two hour intervals Following regularization this patient lost 12 lbs of edema

The most recent drug to be used for paroxysmal ventricular tachycardia is procaine amide (pronestyl) This is given intravenously in doses of about 1 0 gm in a volume of only 5 cc and may be injected within three to five minutes It appears to be safe and have very few untoward effects I have seen it produce prompt reversion in a few minutes This may prove to be the procedure of choice It also can be administered in oral tablets (1 0 to 2 0 gm daily) to prevent recurrences and it is useful in the treatment of ventricular extrasystoles as well It apparently has little beneficial value in supraventricular arrhythmias

DIAGNOSIS OF TYPES OF PAROXYSMAL RAPID HEART ACTION

As an aid in the diagnosis of the various types of paroxysmal rapid heart action a composite figure of the effect of vagal stimulation on these arrhythmias has been constructed (Fig 74) A study of this figure will enable the physician to visualize the results obtained by simple physical examination The first tracing shows the effect of right carotid pressure

in a normal heart There is a gradual slowing and a smooth return to the original rate The second curve shows a similar slowing effect when normal tachycardia is present This patient had an acute coronary thrombosis and the rapid heart rate made me wonder whether he had flutter, in fact the appearance of the electrocardiograms on first glance suggested this diagnosis The gradual slowing of the heart with a smooth return to its original rate eliminated this diagnosis The third case illustrates a typical arrest of an attack of auricular tachycardia One observes the abrupt cessation of the rapid rate with a prompt resumption of normal beats Interruptions during the transition due to extrasystoles or vagal effects are common No other type of rapid heart action is completely controlled in this fashion

The fourth tracing shows the effect of carotid pressure in auricular flutter The ventricular rate is promptly slowed (although the auricular rate remains unaffected), but there follows an ' irregular return to the previous rapid rate The point that distinguishes flutter from normal tachycardia is that as the vagal effect subsides the ventricles return to their original rate in a jerky fashion After a short cycle a longer one may appear, then another short one, until finally the constant rapid rate is resumed This is to be compared with the second tracing where it may be seen that each beat becomes shorter and shorter until the original rate is restored The fifth set of electrocardiograms illustrates the events in auricular fibrillation The rhythm is grossly irregular at the start, becomes slower during the vagal stimulation and then returns to its original absolute arrhythmia The final curves are those of ventricular tachycardia Here no effect whatever is produced Very slight irregularities which are fairly characteristic of this condition continue throughout the tracing

When the changes that have been described are translated into corresponding auscultatory findings obtained over the precordium, it is striking what accurate diagnoses can be made by simple bedside methods

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Acute Cardiovascular Emergencies

A practicing physician is often confronted with apparent or real circulatory emergencies and needs to have some basis for making quick decisions. One of the most important of these decisions is whether or not the situation is serious. A fainting spell may be quite benign or indicative of a very grave disorder. A sudden severe pain may be a simple cramp or a spasm or may be due to an embolus. An attack of suffocation at night may be due to a spasm of bronchial asthma which will shortly leave the patient none the worse or it may be one of acute pulmonary edema. The purpose of the following discussion is to consider some of the more common cardiovascular upsets in which the patient or his family are apt to call the physician suddenly because they believe a serious emergency exists.

Benign Syncope

One of the commonest sudden circulatory upsets is a fainting spell. Simple syncope is not very frequent in organic heart disease. There are many patients with valvular or myocardial disease who never faint and when a brief loss of consciousness occurs it is not always related to the organic lesion found in the heart. The only valvular disease in which syncope occurs with any frequency is aortic stenosis. This complication was discussed previously (Chap 4). Occasionally the onset of any form of paroxysmal rapid heart action (Chap 13) is associated with syncope. In most cases a simple fainting attack is a benign event occurring in otherwise healthy individuals. The precipitating factors for this are varied. The sight of blood, an overheated or crowded room, a sudden fright or the prick of a needle at the time of a vaccine injection or as blood is withdrawn for a Wassermann test, *all* may make some people faint. Another common cause is standing erect and fairly motionless for a considerable length of time. A sudden change of posture from the recumbent to the upright position is another cause of fainting. All these are intimately related to the nervous control of the vasomotor apparatus. In most of these instances loss of consciousness would not have occurred had the individual been recumbent. There is apt to be a splanchnic dilatation and

a temporary cerebral anemia which naturally becomes aggravated if the body is in the upright position For this reason it is customary to lower *the head between the knees to revive one who has fainted*

If a person is seen during a faint it will be observed that he quickly grows pale and at the outset there is a fall in both the heart rate and in the blood pressure Generally these early changes are not noted because by the time a physician arrives the heart rate has risen and the normal blood pressure level has been restored There is much in this upset that resembles a vagal explosion or a temporary hypervagotonic state In fact I recall once seeing a young man who could faint at will After suddenly changing his position from recumbent to upright several times he would always faint For an hour or so after a subcutaneous injection of 2 0 mg ($\frac{1}{30}$ grain) of atropine sulfate, he could not be made to faint It appeared that atropine paralyzed the vagus and prevented the reflex from taking place This need not be the only or even the most common mechanism that controls benign syncope The sympathetic nervous system may be involved in some reflex way that permits changes in blood pressure From a therapeutic point of view it is wise to try tincture of belladonna, 10 drops after each meal or ephedrine sulfate 0 025 gm three times a day when such attacks recur with sufficient frequency to merit medication Wearing a tight abdominal corset may also be of some value It is hardly necessary to recall that sudden massive internal hemorrhage needs to be thought of as a possible explanation of a fainting spell When no organic disease is present, a patient having benign syncope should not be restricted in his activities and he should be assured that his heart is sound

There are frequent instances in which syncopal attacks remain unexplained, even showing nothing abnormal during the attack except for the state of unconsciousness The heart, pulse rate and blood pressure may remain unchanged Subsequent postmortem examination may fail to throw light on the mechanism of such episodes I have seen frequent attacks of unconsciousness of this type in a patient who eventually showed three old coronary thromboses Syncopal attacks may also occur for some unknown reason in cases of cortical atrophy or cerebral sclerosis In these cases one can assume that transient cerebral spasm takes place

Epilepsy

Obviously benign syncope of the type just described needs to be distinguished from petit or grand mal The difficulty is not great if one witnesses the attack for there is no change in the pulse rate in epilepsy I have made the error of confusing the two conditions when judgment had to be made from the history alone The family history of epilepsy, the occurrence of an aura the loss of sphincter control, the occurrence of convulsions and the postparoxysmal headache or sleep may help to differentiate the two It is obvious that cardiac therapy would be of no avail in syncope due to epilepsy but that instead phenobarbital or dilantin would be used

Adams-Stokes Syncope

There is one condition in which syncope is a major and characteristic event and is the result of serious disease of the heart, i e, Adams-Stokes disease Here sudden unexpected painless unconscious spells occur with or without convulsions These attacks are due to a sudden failure of the ventricles to contract or a sudden marked slowing of their rate The severity of the paroxysm will in a large measure depend on the duration of ventricular asystole If this only lasts several seconds, the patient merely feels a light wave" come over him If it is longer he will lose consciousness and fall If it lasts still longer convulsions occur and if the beat is not resumed after a few minutes, death ensues When the attack ends the patient may feel perfectly well and be ready immediately to resume his former state These attacks come suddenly, generally without warning or aura and with varying frequency In some the spells are rare, in others there may be many during the same day, even producing a "status epilepticus

The treatment for the syncopal attacks of Adams-Stokes disease, apart from whatever underlying condition may exist, is ephedrine or adrenalin The former is given in doses of 0 025 to 0 05 gm ($\frac{3}{8}$ to $\frac{3}{4}$ grain) by mouth two or three times a day and the latter subcutaneously in doses of about 0 3 to 0 5 cc of 1 1000 solution Propadrine may be substituted for ephedrine, the dose being the same When the situation seems urgent and immediate effects must be obtained adrenalin should be used and repeated as often as seems necessary, even every two hours in some cases When attacks recur at very rare intervals, it is hard to evaluate any therapy, but ephedrine may be tried by mouth There are rare instances in which adrenalin has been injected directly into the heart during a prolonged period of asystole with recovery of the patient (Chap 21, Fig 104) It is obvious that under such circumstances a subcutaneous injection would be useless as blood flow has already ceased and no absorption would take place There is also suggestive clinical and fairly sound pharmacologic evidence in support of the value of barium chloride (0 03 gm four times a day) in the prevention of recurrent attacks of this sort Other measures that seem occasionally to be useful are inhalations of 1 100 adrenalin solution, full doses of atropine, metrazol intramuscularly or by mouth, thyroid extract and complete digitalization

Ventricular Fibrillation

Another type of syncope that results from a disturbance of the mechanism of the heart beat is one due to ventricular fibrillation Although this is generally a fatal condition and very likely one of the causes of sudden death in patients with coronary artery disease, rarely it is a transient phenomenon When it occurs the ventricles actually stop beating and fail to eject blood into the circulation If contractions are not quickly resumed death results This mechanism is apt to follow a preliminary period of ventricular tachycardia or frequent ventricular extrasystoles

(Chap 21, Fig 103) When it can be established that syncopal attacks are due to such disturbances, quinidine sulfate (0 2 to 0 3 gm three times a day) should be tried, as both from a theoretical and a practical point of view such medication may do away entirely with the attacks or at least *diminish their frequency or severity*

Carotid Sinus Syncope

Quite a different type of syncope that particularly has been studied in recent years is that which results from carotid sinus irritability Soma Weiss and his coworkers discovered a considerable group of people with hypersensitive carotid sinus reflexes, some of whom are prone to fainting attacks It has long been known that pressure over the carotid artery can often slow the heart beat and this mechanism has been used to stop attacks of paroxysmal tachycardia (Chap 13) and to study certain cardiac arrhythmias, especially auricular flutter and heart block It previously was thought that the method of action of this manipulation was by direct stimulation of the vagus nerve which lies beneath the carotid artery It was shown, however, by Hering that this is not the correct explanation, but that the effect is produced by irritation of nerves surrounding the carotid artery at its bifurcation into the internal and external branches which produces reflex effects (carotid sinus reflex) Although under normal conditions the carotid sinus undoubtedly serves an important role in regulating certain bodily functions related to the blood pressure level, the heart rate, the effect of changes in posture and other cerebral phenomena, in some individuals the sensitivity of this region is increased and results in a tendency to dizziness, syncope and convulsions This occurs more frequently in patients with arteriosclerosis or hypertension but may be present without such changes Increased sensitivity of the carotid sinus may also result from local irritation produced by a neighboring gland or a tumor in the neck In some cases syncope is apt to occur as a result of a twist of the neck to one side or the other or to a peculiar position of the head from wearing a high collar

Whenever a problem of syncope arises that is not readily explained by some other cause the patient should be tested for carotid sinus sensitivity The proper part of the carotid artery should be sought for and will generally be identified by the finding of a local bulge or prominence at its bifurcation First light and then more firm pressure and irritation should be applied, preferably with the patient sitting rather than recumbent The pressure should be exerted for several seconds first on one side and then on the other Pressure should not be continued for more than a few seconds at a time because if the carotid circulation is shut off for too long hemiplegia may result When a positive result is obtained the primary complaint of syncope will be reproduced and there is apt to result a fall of blood pressure or a slowing of the heart, although syncope may even take place without these effects When the attacks of syncope can be ascribed to an increased sensitivity of the carotid sinus, it has been shown that the administration of daily doses of ephedrine sulfate (25 to 50 mg

two to four times a day) may be effective in preventing such attacks Occasionally atropine or tincture of belladonna (10 drops three times a day) may also inhibit the attacks On the rare occasions in which a local tumor or gland is responsible for the attacks, removal of the cause is curative Furthermore, Weiss and his collaborators obtained some permanent cures in a small number of cases by resecting the nerve plexus from the carotid artery Finally, Ray and Stewart report some cures in cases of carotid sinus syncope by intracranial section of the glossopharyngeal nerve

Cerebral Accidents

Sudden prolonged unconsciousness is commonly due to cerebral hemorrhage or thrombosis, subdural hematoma or cerebral embolism With all these there is apt to be paralysis of one side of the body and if the speech centers are involved aphasia also results It is important to realize that cerebral hemorrhage occurs in older persons with hypertension When a sudden hemiplegia develops with or without unconsciousness in one who has not a significant hypertension there are several conditions to bear in mind One must think of syphilis of the brain, polycythemia, Buerger s disease and brain tumor When sudden paralysis occurs in younger people, especially when ocular muscles are involved, a rupture of a *small intracranial aneurysm* must be suspected Such aneurysms are thought to be due to congenital deformation and not to syphilis Finally embolism to the brain may cause sudden unconsciousness or hemiplegia Some evidence of previous heart disease will then be discovered It occurs mainly under four sets of circumstances—mitral stenosis, auricular fibrillation, cardiac infarction and bacterial endocarditis When it is associated with the first two an embolus becomes dislodged from a sterile mural thrombus in the left auricle When it develops from the third condition the embolus arises from a mural thrombus in the left ventricle that resulted from an infarction of the musculature following a coronary thrombus Sterile mural thrombi may be present in the cardiac cavities for years without doing any harm and at present there is no way of detecting their presence or of predicting whether embolism will result In bacterial endocarditis septic emboli are dislodged from the vegetations on the affected valves

There is one aspect of cerebral embolism which has not been explored but which may be of interest It has impressed me that most patients with mitral stenosis complicated by cerebral emboli have little or no dyspnea In general they have fairly good function and have been able to lie quite flat in bed On the contrary, cerebral emboli are very rare in mitral cases with advanced cardiac failure or with orthopnea One wonders whether or not the upright position of the neck and head makes it more difficult for the small thrombus, dislodged in the blood stream, to go up the carotid arteries If the clot is heavier than blood it would remain in the lower portion of the stream and spare the cerebral circulation Other possible explanations are that with advanced failure the velocity of blood flow is decreased and this would tend to diminish the likelihood of

emboli or that emboli are dislodged soon after mural thrombi develop If the latter is true it would account for the occurrence of emboli early rather than late in the progress of mitral stenosis or auricular fibrillation This problem needs further investigation

It has been stated that cerebral hemorrhage occurs with hypertension It is important to realize that the elevation in blood pressure is very apt to persist after the rupture of the cerebral vessel even if the patient is unconscious Therefore, one should hesitate to regard the condition as due to cerebral hemorrhage if the blood pressure is normal It may be found to be due to one of the other causes previously mentioned At times one is thereby led to suspect the presence of a mural thrombus in the heart or cardiac infarction Finally, sudden paralysis of one side of the body may take place only to disappear in a few hours This may even be repeated I have seen complete temporary hemiplegia recur several times during the course of a week in a man with hypertension This very likely is due to transient spasm of the cerebral arteries and not to any permanent occlusion or rupture of a vessel It is clear, therefore that sudden cerebral accidents present a problem in differential diagnosis and that they should not be too readily regarded as instances of an ordinary apoplectic stroke

The prognosis for patients having these cerebral accidents will naturally vary considerably These hemorrhages are almost never instantly fatal In this respect they differ from coronary attacks, which often kill instantly When coma develops and continues for more than a day or so the outlook is grave A slight fever and leukocytosis are common findings and are merely the result of the cerebral infarct or of the extravasated blood A hypostatic bronchopneumonia is a frequent complication during the early days following a cerebral accident in elderly people and is apt to prove fatal if not treated If the patient survives the first week he generally recovers When paralysis has occurred some recovery of function is the rule and on the whole improvement in the power of the leg is greater than that of the arm In some, although physical recovery may be satisfactory permanent mental impairment may result

If the diagnosis of cerebral embolism is made promptly it may be worth while to try an injection of the stellate ganglion with procaine Some prompt beneficial results have been reported Inasmuch as this is a fairly simple procedure and there is little else that one can do therapeutically it seems justifiable

The treatment for cerebral vascular accidents is supportive Occasionally lumbar puncture is performed or hypertonic salt solution or magnesium sulfate is given by mouth by rectum or intravenously to diminish intracranial pressure The results from these methods of treatment have not been very impressive and the same may be said of phlebotomy If the spinal fluid is bloody or it is under increased pressure repeated punctures may be helpful Caffeine sodium benzoate given intramuscularly in full doses (0 5 gm) may be useful as a respiratory stimulant and digitalis, if there are indications for its use such as auricular fibrillation or congestive

heart failure When a localized subdural hematoma is suspected trephining the skull or exploratory operations would be indicated At times x-ray examination may reveal dislocation of the pineal gland and thereby guide the surgeon to the correct side for operation Even exploration of both sides of the brain may be necessary to find the localized blood clot

Paroxysmal Dyspnea

A common cause for sudden alarm and one for which the physician may be hurriedly called is a paroxysm of dyspnea or palpitation Paroxysmal dyspnea is most apt to occur at night and is often associated with Cheyne-Stokes breathing It is common in hypertensive heart disease, in luetic aortic insufficiency and in disease of the coronary arteries It occurs much less frequently in rheumatic valvular disease The patient is awakened from sleep with suffocation and air hunger He is agitated and develops a cough and a wheeze The typical picture of acute pulmonary edema with abundant pink frothy sputum may follow The patient often has a cold sweat and struggles for air The attack when mild may last only fifteen minutes to a half hour and be followed by a fairly comfortable sleep When it is more severe the dyspnea continues uncontrolled and the patient s life seems to be at stake It is generally wise for the physician to administer a hypodermic injection of 0 010 to 0 015 gm ($\frac{1}{6}$ to $\frac{1}{4}$ grain) morphine and 0 6 mg ($\frac{1}{100}$ grain) atropine Within fifteen minutes after such treatment most of the attacks subside Occasionally a phlebotomy of 500 cc is extremely valuable This is more beneficial if hypertension is present The same result may be approximated by applying tourniquets to the extremities, thereby producing peripheral venous stasis After the acute emergency is over adequate digitalis should be given to control future attacks Another therapeutic measure that is often effective is the intravenous injection of aminophylline (0 24 to 0 48 gm) This may stop an attack in five to ten minutes It is also valuable in spells of acute bronchial asthma

Paroxysms of dyspnea occur in still other conditions and for other causes than those just described Occasionally dyspnea and even acute pulmonary edema is the initial event in acute coronary thrombosis and treatment will then have to be directed accordingly A patient may have a pulmonary embolism or infarct of the lung and be taken with sudden breathlessness and generalized pulmonary edema It must be remembered that hemoptysis or bloody sputum frequently is absent in these conditions Furthermore, a sudden increase in heart rate due to paroxysmal rapid heart action of one form or another may quickly produce air hunger, especially if it develops in one who already has significant organic cardio vascular disease The differential diagnosis and treatment for these paroxysms have already been discussed (Chap 13) Suffice it to say that such disturbances in the mechanism of the heart beat must be sought for because the proper treatment, which is generally very effective, will depend on accurate diagnosis In the ordinary case of paroxysmal dyspnea (so-called ' cardiac asthma) the heart rhythm remains normal and the rate

only slightly accelerated In these other conditions the rate is very rapid and the rhythm may be either regular or irregular Some patients will require large doses of digitalis, others may be readily controlled by pressure over the carotid sinus or the eyeball and a third group will require *quinidine therapy*, depending on whether the paroxysm is due to auricular fibrillation or flutter in the first instance to auricular tachycardia in the second or to ventricular tachycardia in the final instance

Pulmonary Embolism and Infarction

Sudden dyspnea or pain in the chest or both may be due to a pulmonary embolus The condition may closely simulate an attack of coronary thrombosis The clinical features of the latter have already been discussed (Chap 6) and need not be repeated A large pulmonary embolus may cause death within five or ten minutes It rarely kills as instantly (in seconds or a minute) as does a coronary attack Frequently the agony lasts longer and in many instances recovery occurs Infarction of the lung need not take place during the early minutes or hours of such an attack as a longer time is required to produce these changes The clinical features will vary considerably depending on the size of the pulmonary embolus and upon the suddenness of the occlusion It must also be borne in mind *that pulmonary thrombosis can occur because of local changes in the lung* that is congested and is not always due to embolism

The most alarming and sudden cases are due to emboli dislodged from peripheral veins, especially those of the leg and the pelvis Such venous thrombosis is common after surgical operations on the abdomen and embolism may occur whether there has been clinical evidence of phlebitis or not These accidents are most common about ten days postoperatively but may develop any time Attention has recently been called to the fact that fatal pulmonary embolism may result from deep seated phlebitis following apparently innocent traumatic injuries of the legs In patients with organic heart disease pulmonary embolism follows the dislodgment of bits of mural thrombi from the right auricle especially when mitral stenosis or persistent auricular fibrillation has been present

Pulmonary embolism or thrombosis may cause sudden dyspnea with or without chest pain The breathing may have a peculiar character, as if the patient has a foreign body in his throat—a gasping struggle with the mouth widely open, like a fish out of water but very little air is inspired When pain is present it rarely has the radiation to the arms that characterizes coronary thrombosis Cyanosis is also variable Sudden faint feeling or actual loss of consciousness without any pain is a common initial symptom Frequently it will be observed that the pulse and temperature had been rising for a few days before the more dramatic acute manifestation of pulmonary embolism took place These premonitory features are probably due to the underlying thrombophlebitis The picture of shock is frequently present as in coronary thrombosis If the patient survives the first twelve hours, fever and leukocytosis develop When infarction of the lung results hemoptysis can occur Bloody sputum,

heart failure When a localized subdural hematoma is suspected trephining the skull or exploratory operations would be indicated At times x ray examination may reveal dislocation of the pineal gland and thereby guide the surgeon to the correct side for operation Even exploration of both sides of the brain may be necessary to find the localized blood clot

Paroxysmal Dyspnea

A common cause for sudden alarm and one for which the physician may be hurriedly called is a paroxysm of dyspnea or palpitation Paroxysmal dyspnea is most apt to occur at night and is often associated with Cheyne Stokes breathing It is common in hypertensive heart disease, in luetic aortic insufficiency and in disease of the coronary arteries It occurs much less fre [illegible] matic valvular disease The patient is awakened fro [illegible] tion and air hunger He is agitated and develop [illegible] The typical picture of acute pulmonary edema [illegible] thy sputum may follow The patient often ha [illegible] or air The attack when mild may last [illegible] and be followed by a fairly comfortable [illegible] dyspnea continues uncontrolled and the [illegible] It is generally wise for the physician to [illegible] of 0 010 to 0 015 gm ($\frac{1}{6}$ to $\frac{1}{4}$ grain) [illegible] atropine Within fifteen minutes after [illegible] subside Occasionally a phlebotomy of [illegible] This is more beneficial if hypertension is [illegible] be approximated by applying tourniquets to the extrem [illegible] oducing peripheral venous stasis After the acute emergency [illegible] adequate digitalis should be given to control future attacks Another therapeutic measure that is often effective is the intravenous injection of aminophylline (0 24 to 0 48 gm) This may stop an attack in five to ten minutes It is also valuable in spells of acute bronchial asthma

Paroxysms of dyspnea occur in still other conditions and for other causes than those just described Occasionally dyspnea and even acute pulmonary edema is the initial event in acute coronary thrombosis and treatment will then have to be directed accordingly A patient may have a pulmonary embolism or infarct of the lung and be taken with sudden breathlessness and generalized pulmonary edema It must be remembered that hemoptysis or bloody sputum frequently is absent in these conditions Furthermore a sudden increase in heart rate due to paroxysmal rapid heart action of one form or another may quickly produce air hunger, especially if it develops in one who already has significant organic cardiovascular disease The differential diagnosis and treatment for these paroxysms have already been discussed (Chap 13) Suffice it to say that such disturbances in the mechanism of the heart beat must be sought for because the proper treatment, which is generally very effective, will depend on accurate diagnosis In the ordinary case of paroxysmal dyspnea (so called cardiac asthma) the heart rhythm remains normal and the rate

of the limb and disappearance of the arterial pulsation usually means an embolus Many physicians still confuse arterial occlusion and venous phlebitis In the latter condition the limb is not cold and there eventually develops swelling, whereas in the former there is no swelling and the *temperature of the affected part falls* Some patients with embolism recover on symptomatic treatment Embolectomy together with heparin therapy has been practiced at times with excellent results and at other times with out success It is best performed during the first six hours after the onset The use of an apparatus which produces artificial alternate suction and compression of the limb may be of aid in this problem and make embolectomy unnecessary When gangrene has already developed in a limb, am putation will be necessary Newer methods are being tried such as procaine hydrochloride injections in the lumbar sympathetic system and freezing the limb involved

Paroxysmal Rapid Heart Action

Any of the forms of paroxysmal rapid heart action discussed in the previous chapter may be the cause of a cardiovascular emergency When organic heart disease already is present, though compensation may be excellent or even if the heart is structurally normal, a sudden change of rate from 80 to 160 or 200 may produce a state of collapse or shock or sudden acute pulmonary edema At times with the onset of such a rapid rate actual syncope may take place and in some cases anginal pain may occur The physician should make every effort to examine the heart during the attack in order to establish the exact type of abnormal rhythm that is responsible for the treatment (see Chap 13) *will vary with the different* forms

Acute Hemorrhage

Many instances of acute hemorrhage have obvious causes and present an emergency situation A massive nosebleed, a large hemoptysis or hematemesis presents individual problems Treatment is then directed at the underlying condition whether it is hypertension pulmonary tuberculosis, cirrhosis of the liver or peptic ulcer Occasionally with extensive internal bleeding diagnosis may become very difficult Hypertensive pa tients may have acute hemothorax or hemoperitoneum Of particular interest are cases of acute and massive gastrointestinal bleeding Because of the sudden shock and collapse these patients may resemble those with coronary thrombosis Only in a day or so, when a dark or tarry stool is passed, may the true nature of the disease become apparent Furthermore, during the state of shock accompanying severe hemorrhage coronary thrombosis occasionally develops as a *complication This increases the* difficulty in differential diagnosis

It can be readily seen from the foregoing discussion that the emergencies in cardiovascular disease are numerous Sudden events and changes in the clinical condition of the patient are found to have a lim ited number of causes They require a mechanism that can produce a

sudden alteration in the blood supply of an individual part of the body or that suddenly changes the mechanism of the heart beat A blood vessel can rupture or become partially or completely occluded The heart may suddenly take on too rapid or too slow a beat as a result of a disturbance in the normal rhythm Finally, certain sudden nervous influences or reflexes may be set at work producing vasomotor changes or alterations in the blood pressure resulting in syncope They all require individual analysis, for without this, intelligent treatment cannot be carried out

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Medicolegal Aspects of Heart Disease

Physicians have become more and more concerned with the relation of trauma to heart disease The decisions that need to be made by courts, industrial accident boards and insurance companies often require medical opinions as to the existence of heart disease or the possible development or aggravation of preexisting heart disease following trauma or accidents We physicians have had little to do with the formulation of the existing laws and may believe that some are imperfect or even unwise That is not our present concern It is expected that we should offer intelligent and honest opinions as to the medical problems involved, leaving it to the judge and jury to interpret the law in the light of these facts and opinions

As an illustration of a situation that seems unfair or at least unwise, the following may be mentioned Two men were injured in the same way while walking and died several months following the accident, as a result of the injuries received One was a young man 25 years of age who was perfectly well, and the other was a man 68 years of age who was known to have had hypertension and angina pectoris In the first instance the young man s life expectancy was shortened forty years or more, while in the second it was shortened possibly two or three years In the eyes of the law in many courts or industrial accident boards the damages for the death of the two individuals would be identical It would seem more just from a sociologic point of view to differentiate the total amount of harm done to an individual by a specific accident according to the life expectancy, when a known organic condition exists This and other aspects of the problem are making it difficult or impossible to obtain employment in industry for patients with heart disease, even when the cardiac abnormality is trivial

There are many circumstances in which a physician is unable to answer questions of cause and effect if heart problems arise after accidents The experience of most of us is confined mainly to the ordinary progress of heart disease Our knowledge concerning the effects of trauma on the circulation is limited Because of this lack of knowledge, the honest physician is often compelled to say he does not know whether a certain blow to the leg or chest or a fright can cause myocardial injury or any given arrhythmia He may reason to the best of his ability and decide that the particular abnormality could not be or was a very unlikely result of the accident [illegible]

might state that the cardiac condition was a coincidence or was present before the accident, or was the result of the natural progress of disease He would be influenced by his limited theoretical knowledge and by the fact that he had not associated such a causal relationship in his general practice Similar points of view have been taken in the past concerning certain matters, only to find that years later such opinions were wrong A good example of this is pain in the back following severe injuries When such pain continued for months or years and physical examination includ ing x ray studies revealed no abnormality, the condition was often called neurosis or 'railroad spine In some cases it would even be inferred that the patients were malingering No doubt many of these cases were functional or neurogenic but we know now that some were due to a dislocated and compressed intervertebral disk Only in recent years has this condition been recognized

The same situation exists in relation to heart disease Formerly it was difficult to understand how a patient might develop angina pectoris or symptoms of coronary artery disease following trauma If a man complained of anginal symptoms after an accident, I previously took the position that he must have had symptoms of this before but was not telling about them The most that one could admit was that the condition was aggravated by the accident Through the interest of Warburg in Denmark and Beck and Beas in this country, we have learned that direct blows to the chest, even without fractures of ribs or actual abrasion of the skin, can traumatize the heart muscle or coronary arteries so as to produce symptoms of coronary insufficiency The steering wheel' accidents that are so common now are good examples of the type in which contusion of the heart muscle occurs There are now well authenticated cases in which after an accident there developed definite angina pectoris or coronary thrombosis Even instances of rupture of the heart or of a valve have been observed These cardiac conditions not only may result from direct trauma of the chest, but also from severe injuries to other parts of the body or as a result of very severe and unaccustomed strain I recall seeing an instance of a ruptured mitral valve which resulted from the severe strain of rowing a boat in a storm The ruptured valve was found on postmortem examination In this case increasing dyspnea and congestive failure developed, the man dying several months after the strain

Of considerable importance from a medicolegal point of view is the dif ferentiation of pulmonary embolism and coronary thrombosis This prob lem has already been discussed in Chapter 6 It may be emphasized that after traumatic accidents, especially those involving fractures or bruises of the legs and the application of casts, thrombophlebitis and subsequent pulmonary emboli are common and that they are often misdiagnosed as posterior myocardial infarction Occasionally disinterring the body even months after death has clarified the situation (See pp 117-118)

Arrhythmias of the heart may also result from unusual physical or mental strain Many such cardiac irregularities produce no significant ill effects Others are of more importance I remember seeing a man who

developed auricular fibrillation directly after falling down an elevator shaft In another case, a man who had been operated upon for hyperthyroidism and had been symptomatically cured had a recurrence immediately after being held up in the street by a burglar The symptoms of hyperthyroidism with auricular fibrillation promptly returned

The most important point in many of these cases is the time relation between the accident and the cardiac abnormality If the exact status of the patient before the accident is known, it is reasonable to assume that new objective or subjective evidence of disability occurring within minutes, hours or several days is due to the accident Objective evidence is obviously more reliable than subjective It, therefore, is important to record findings such as heart rate and rhythm, blood pressure, electrocardiograms the presence of rales in the lungs, x rays, and so on, as soon as possible after an accident When the physician has to depend on symptoms, due regard must be had to functional and emotional reactions and to the possibility of malingering However, the courts and some physicians have had a tendency to minimize the importance of post traumatic neurosis Many of the soldiers of the First World War who suffered severe neurocirculatory asthenia have ever since been much more handicapped than others who had fractures of bones or gunshot wounds of the abdomen or chest In some of these cases the nervous symptoms have continued indefinitely, even when the question of war compensation did not enter into consideration This has been found to be true in a follow up study in England and has its counterpart in civil practice

The following experience is illustrative A man of 40 who had always been well and strong had worked steadily for many years He had been accustomed to manipulate very heavy steel beams One day he was hit by a swinging beam and sustained a violent crushing blow to the body An arm and ribs were fractured and it took a few months for physical recovery When the patient became ambulatory he complained of weakness, palpitation, dyspnea on effort and tremulousness These are the classical symptoms of neurocirculatory asthenia I found no evidence of organic heart disease or any other significant organic disease I regarded this working man as being in a worse condition than if he had [illegible] with a permanent limp in a leg or a contracture of an arm, but without any nervous symptoms I felt certain that he would never be able to do the strenuous physical work to which he was formerly accustomed I am afraid that such patients, when we feel that they are honest, receive poor treatment in medicolegal controversies

The time relation is also very important when the question of aggravation of a preexisting condition comes up If a new chain of [illegible] or findings develops a few hours or a few days after an accident and the clinical status before the accident appears to have been stationary, it is fair to assume that some degree of aggravation occurred On the other hand, if examination reveals that there was no change for a few weeks, and certain disabilities resulted thereafter, it is extremely [illegible] that the accident was the cause of the change In a case of this sort a very minor

accident was supposed to have aggravated a preexisting mitral stenosis and auricular fibrillation I had reliable data before and after the accident which showed no change whatever The patient claimed his breathing was worse, but I knew that the vital capacity of the lungs was in no way affected This enabled me to maintain that the state of cardiac efficiency was not altered by the accident

The main difficulty is in interpreting symptoms When the problem of angina is involved, we are so dependent upon the subjective complaints that errors can easily be made, either for or against the interest of the patient We must first be certain of the diagnosis and then we should try to get some proof that the anginal pain is more frequent, more severe, of greater duration or brought on by less effort If there is reason to doubt the veracity of the patient, he may need to be 'shadowed' in order to check his statements This does not come within the province of a physician as we are not detectives but rather guardians of the health of our patients It may be the work of a lawyer or the insurance company In all these matters scrupulous care and honesty on the part of the examining physician are paramount

Finally, the troublesome question of 'total and permanent disability" will be discussed From a purely sociologic point of view, this provision in insurance policies has often proved to be a great blunder It has undermined the morale of many an honest citizen and has forced some physicians into practices which, from an ethical point of view, are at least open to question Many of us would be happier if no such insurance policies had ever been written Insurance companies are suffering tremendous losses which must be borne by all policyholders This is partly due to the unexpected increase in chronic cardiovascular disease that has taken place during the past decades and partly to the economic depression of previous years Added to this is the fact that many unrighteous claims have been granted because of the dishonesty of policyholders, occasionally with collusion of unscrupulous physicians

The first difficulty comes in interpreting the term total and permanent disability ' Some totally blind or deaf people support themselves A man could be bedridden and yet earn something reading proof or translating foreign medical publications, as I know one physician did for two years It is the duty of the court to make a legal interpretation of this phrase When a person takes out such insurance, he probably has in mind a protection against his inability to continue his accustomed work If a man is an active obstetrician and has to climb stairs to get to his patients' homes, and must be able to exert himself vigorously when using forceps, then increasing breathlessness from cardiac weakness or from emphysema of the lungs renders him unfit to continue his occupation permanently However, if he is a dermatologist with mainly an office practice, he may be able to carry on for some months or years longer Now, are we to say that one or both are totally and permanently disabled? Many a disabled individual could carry on some mild or sedentary occupation, if he were trained differently and if such a position were available But we cannot

make a dermatologist out of an obstetrician overnight Likewise a day laborer cannot easily be trained to be a cashier The best we as physicians can do is to express an honest opinion as to whether or not a given occupation is an added hazard to the comfort or life of the patient concerned

It is not our duty to help insurance companies undo losses that they have sustained If we make an unwise investment in a home or in business, we have to suffer the loss Insurance companies made what have proved to be unwise contracts and must inevitably pay for their mistake On the other hand, we must protect them against any false claims, by a most painstaking medical analysis, keeping in mind the interest of the patient and the company Unfortunately, the final decision may depend on the amount of insurance involved If a physician in general practice has insurance protection of $4000 a year against total and permanent disability, his position may be different if he earns $10,000 a year than if he earns only $4000 yearly When he develops angina pectoris and finds it hard to climb stairs or drive his motor car in wintry weather he may well prefer to carry on because of a greater income, even if he may shorten his life or have more attacks of angina If his income is no greater than his insurance, he is much less likely to want to suffer any more than necessary A physician may quite reasonably urge one man to carry on and another to quit work when the physical disability is the same in both cases At times it seems that the advantages of retiring are not great enough to warrant the financial loss involved In some cases the joy and satisfaction of working compensates for the increase in discomforts Some physicians would choose to have more attacks of angina but continue their practice I believe that a man has a right to choose whether he will have more suffering and keep at work, or less suffering and accept his insurance protection provided there is no doubt about the diagnosis and reality of the symptoms or disability

There has been a move on the part of some insurance companies in very recent years that seems wise and should be extended When a patient has coronary artery disease and his condition seems fair, it may be advisable to permit him to try to resume some work If such an individual has been receiving total permanent disability benefits, some companies will give him a trial period of three months, during which time full compensation is continued while he resumes some or all of his duties If at the end of that time it is clear that he cannot carry on, he again retires and continues to receive disability payments If his progress is favorable after this three months trial period, he then discontinues receiving the disability benefits and carries on with his work This is a wise and useful provision, both for the insurance companies and the policyholders, for it may save the former expense and may help to rehabilitate the patient Physicians should cooperate in this plan with the understanding that the insured will not jeopardize his interests by making an honest effort to return to work

To return to the difficult problem of angina in which subjective complaints are so important, it may be necessary to perform special tests for

diagnostic purposes It also may be necessary to "shadow" the patient to find out whether he actually is unable to walk or work It must be remembered, however, that detecting a man walking a mile or two in Florida in the winter does not necessarily mean that he can walk one block in New England There are many individuals who can exert themselves a great deal in warm climates who can do very little in cold ones One should try to check the statements that are made in direct relation to the individual's ordinary working activities Furthermore, it is fair for the physician to assume that his patient is honest, until he knows otherwise This is particularly true when the family physician has known the patient for many years and has always found him to be honorable I have known instances in which men were receiving insurance for total disability which I thought was entirely unjustified, and I have informed the companies to that effect Unfortunately, too, I have seen instances when patients were disbelieved, had their insurance discontinued, only to prove their honesty by dying of a coronary thrombosis The physician has a difficult task Only by great care and wise and honest regard both for the interest of our patients and the protection of insurance companies, can the high standards of our profession be maintained and the services expected of us be rendered

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The Significance of Bronchial and Other Factors in the Production of Dyspnea

Breathlessness is the most important symptom of heart disease In the early stages of cardiac failure it may be the only evidence that the cardiovascular apparatus is inefficient This is true whether the problem is one of valvular or nonvalvular heart disease The physical findings at this time may be entirely negative or may be very insignificant, showing only those alterations which are commonly present in patients without any heart disease It becomes very important, therefore, to be able to interpret intelligently the significance of this primary complaint—shortness of breath There are other factors beside heart failure that play their respective roles in the production of dyspnea and some of these, especially the bronchial, deserve consideration

Inasmuch as heart disease is extremely common and asthmatic bronchitis is also very prevalent it is not surprising that the two conditions are frequently present in the same individual From a purely statistical point of view it would be expected that a considerable number of people might suffer from both diseases In point of fact, however, I have seen so many patients with organic heart disease, especially rheumatic valvular disease, also suffering from asthma that I have been led to believe that the relationship is not merely coincidental It may be that the prolonged pulmonary stasis of itself which accompanies heart failure in some way renders the bronchial tree more susceptible to the asthmatic state Another relationship which seems to be important is that other allergic stigmata such as a previous history of hay fever, urticaria or chronic eczema or a family history of such diseases are frequently detected in rheumatic cardiac patients One may suspect that such individuals are allergic in two different senses In the first place they are sensitive to the proteins that bring about bronchial asthma, hay fever and urticaria In the second place they may be regarded as allergic in the sense that many manifestations of rheumatic fever are now looked upon as allergic reactions These considerations may explain the undue frequency of asthmatic bronchitis in cardiac patients

Bearing in mind that shortness of breath is the outstanding complaint in both conditions, it is most important to distinguish how much of the

disability is due to each, for the prognosis and treatment will depend a great deal upon this distinction This differentiation is obviously most important because although dyspnea due to asthmatic bronchitis is distressing, it comes and goes and is compatible with a long and fairly useful life, whereas when it is the result of cardiac failure, especially when it is of the paroxysmal type, the outlook is very grave Furthermore, digitalis is helpful for cardiac dyspnea and useless for bronchial dyspnea, and medication like ephedrine, adrenalin and potassium iodide which often is beneficial for bronchial dyspnea is practically of no avail in cardiac failure

Such problems are best illustrated by actual practical experiences Some years ago I was called to see a woman 60 years of age who was suffering from attacks of dyspnea, especially at night She was the mother of a physician and had been seen by a most competent consultant who regarded the condition as hypertensive heart disease with parosysmal nocturnal dyspnea or so called cardiac asthma' This diagnosis carries with it a very ominous prognosis for such patients do not live on the average more than a few years I found that the dyspnea was only of several weeks' duration Physical examination showed the customary amount of arterio sclerosis for a woman of her age There was slight hypertension, the readings being 180 mm systolic and 100 mm diastolic The heart was slightly enlarged, the action was regular with a rate of 90 and there was a slight systolic murmur at the apical and basal regions There was no peripheral pitting edema and no hepatic or venous engorgemnt So far, the findings were compatible with the diagnosis previously made, for many patients with paroxysmal cardiac dyspnea show very little of significance on exam ination, except cardiac enlargement The signs in the lungs, however, gave the clue to the proper diagnosis Rales were heard generally distributed throughout both chests and were readily audible over both sides anteriorly They were inspiratory and expiratory and of the squeaking type such as are found in asthma or emphysema There were no moist rales at the bases of the lungs When rales are due to cardiac failure they are almost invariably moist and inspiratory and limited to the bases of the lungs When they are generally distributed as occurs in acute pulmonary edema they are also moist or bubbling, may occur in both phases of respiration, but the patient is then obviously in an acute serious state Such was not the case here The conclusion, therefore, was that the patient although elderly, hypertensive and somewhat arteriosclerotic, had no heart failure whatever and that the dyspnea was entirely due to asthmatic bronchitis Subsequent developments confirmed this opinion The digitalis she was taking was omitted and instead she was told to take 10 drops of saturated solution of potassium iodide three times a day, $\frac{3}{8}$ grain of ephedrine morning and night, and to use steam inhalations Either as a result of the medications or because of the changes in weather which often have an effect on an asthmatic state she quickly improved and remained ambulatory for many years Congestive heart failure never developed although she lived more than ten years after this experience occasionally having recurring bouts of bronchial dyspnea

The above experience illustrates the misinterpretation of the cause of

dyspnea in a patient who proved to have no element of cardiac failure whatever The following case illustrates the importance of differentiating the various factors involved when there is known organic heart disease This patient was a man about 40 years old who had been coming to the heart clinic for some years He had rheumatic mitral stenosis and auricular fibrillation There was a past history of hay fever He had remained fairly well compensated, taking 0 1 gm of digitalis daily, and was able to attend his little grocery shop One day he was taken into the hospital and was found to be desperately sick He had extreme dyspnea the heart rate was accelerated and grossly irregular, there was a fever of 101 F and he was irrational Extreme dyspnea and orthopnea were the outstanding features His condition was regarded sufficiently serious so that he was put on the dangerous list What amazed the physicians in attendance and comforted the family was that I held out a fair hope for his recovery I did so because a good bit of the respiratory distress could be accounted for on the basis of bronchial dyspnea Although there were many moist congestive rales at the bases of the lungs, there were also numerous asthmatic squeaks throughout both lungs He not only recovered but was able to continue at his light occupation for several years I have had numerous other similar experiences in which the proper interpretation of the various factors producing dyspnea served as the main basis for offering a better prognosis than might otherwise have been made and in instituting more intelligent treatment

The detection of a bronchial factor in cardiac patients is of considerable importance in treating ambulatory cardiac patients in an out patient clinic Here we have a group of individuals belonging to the lower economic strata of life and if they are gainfully employed it is imperative to keep them so whenever possible Picture a married man 40 years of age who has mitral stenosis and auricular fibrillation who works indoors as a salesman and is able to carry on fairly satisfactorily The occupation is not laborious and he manages to keep on the job and support himself We all know that if such a man takes sick leave very often he will lose his job and that he may never be able to find another He has been taking digitalis constantly and thereby the heart rate is kept fairly slow Under these circumstances there will come a time when congestive failure will supervene, requiring a period of rest in bed This will generally be ushered in by increasing dyspnea On the other hand if the increase in dyspnea were due to asthmatic bronchitis we would not only be justified in permitting this man to keep at work but we probably would advise him to do so, in order that he might not jeopardize his earning power Furthermore, increasing the dose of digitalis in these cases is generally of no avail, while a combination of ephedrine and potassium iodide often improves the condition A familiarity with the bronchial factor in the production of dyspnea and the detection of the asthmatic type of rales have formed the background for the proper treatment of these patients This has enabled patients to continue at work who would otherwise have been put to bed and has saved many of them their jobs

There is a serious form of dyspnea really bronchial in origin, which

results from chronic emphysema of the lungs (chronic cor pulmonale). This generally is a slowly progressive process maturing into its severe stage later in life. Occasionally, however, it may appear in its advanced form at middle age. The chest is increased in its anteroposterior diameter, cyanosis develops early and may become quite marked and there is progressive dyspnea. The breath sounds are diminished in intensity, expiration is prolonged, cardiac dullness diminishes because of the overlying expanded lungs and the heart sounds become distant. The intercostal spaces are increased, the diaphragm is depressed and liver dullness is diminished. In a pure case there is right ventricular enlargement. Electrocardiograms often show right ventricular preponderance and complexes of low amplitude. An x ray of the chest may show a prominent pulmonary conus. Objective signs of congestive heart failure, like pitting edema and enlarged liver, come late in the disease. There often is a marked reduction in the vital capacity of the lungs because they are already fully distended and the enlarged chest cavity cannot expand much more. What is even more important is that the entire respiratory cycle lasts much longer than normal and it is difficult to increase the number of breaths per minute. This form of dyspnea and cardiac failure responds very poorly to digitalis but may be helped by ephedrine. The use of morphine is dangerous, especially when the condition is advanced and the vital capacity of the lung is very low. The condition can be ameliorated by a properly fitted abdominal belt. This belt can actually elevate the diaphragm and thereby aid its mechanical movements, increase the vital capacity of the lungs and improve the breathing. Inhalation of oxygen may be helpful but must be administered with caution. In some severe cases oxygen therapy may precipitate coma within fifteen minutes by improving the oxygen content of the blood, which thereby decreases the stimulus to respiration. The result is that in some cases ventilation decreases and gradual coma ensues. The implication is that whenever oxygen therapy is administered in dyspneic states it should be discontinued promptly if the clinical condition of the patient is seen to deteriorate. It is quite striking that in the early stages and often for many years patients with chronic emphysema may be quite short of breath on effort and yet have no orthopnea, being able to lie flat quite comfortably.

A much neglected and overlooked group of cases is one in which there is emphysema and a mediastinal goiter. The latter may be benign or toxic and generally has been present for many years before it is detected, because no enlargement of the thyroid gland need be made out on ordinary physical examination and there may be little if any evidence of thyrotoxicosis. Such cases have increasing breathlessness and often display a peculiar suffused appearance of the face. Roentgenologic examination will reveal the thoracic goiter and often will show a compressed or deviated trachea. If discovered early enough, operation may afford great relief or at least arrest the progress of the condition.

Closely related to pulmonary emphysema is a condition in which the primary pathologic process is in the fine pulmonary arteries. It has been

called Ayerza's disease, the patients with it, black cardiacs. Although it was first thought to be syphilitic in origin most of those cases now recognized have been found to be nonluetic. The striking feature is the intense cyanosis with increasing severe dyspnea and weakness. This is a type of chronic cor pulmonale similar in its manifestations to the severe forms of "emphysema heart."

Patients with advanced pulmonary arterial disease often suffer syncopal attacks. In the late stages peripheral edema may be present. Examination of the heart may show little abnormal except for an accentuated pulmonary second sound. The x-ray reveals evidence of right-sided enlargement and a dilated pulmonary artery. The electrocardiograms will almost invariably display complexes indicative of right ventricular enlargement. Catheterization studies by Dexter have shown that pressure in the pulmonary artery and right ventricle is considerably increased but is normal in the left auricle and in the pulmonary capillaries. These circulatory findings are similar to those obtained in compensated well marked mitral stenosis except that in the latter "pulmonary capillary pressure" is increased. It is of significance that in marked pulmonary vascular disease exercise does not produce an increase in cardiac output.

There are several other factors to be considered in interpreting breathlessness, such as anemia, functional dyspnea and obesity. The first of these can quickly be dismissed. An appreciable degree of anemia is rarely of importance in patients who have cardiac failure from organic heart disease. Occasionally dyspnea is present solely as a result of anemia in patients whose cardiovascular apparatus is essentially normal. Moreover, appreciable dilatation of the heart may also be caused by anemia. Such patients may be erroneously treated for heart disease when liver or iron properly administered is all that is needed. It must be borne in mind that when the hemoglobin content of the blood is sufficiently reduced, dyspnea, especially on effort, can develop because of the diminished oxygen-carrying power of the blood. It is needless to say that digitalis cannot correct this defect. Furthermore, if the anemic state and dilatation of the heart persist for a long time, irreversible cardiac hypertrophy may take place.

A more important problem is dyspnea due to neurotic or functional factors. This frequently takes the form of "sighing breathing" and has been discussed in Chapter 12 and need not be gone into in detail here. Let it suffice to mention that it is common in those who have no organic circulatory disease and is not at all rare even in those with definite structural changes. When this condition is marked and maintained it may result in disturbing hyperventilation. Symptoms of tetany develop with tingling and numbness of the extremities. It is not difficult to interpret properly this type of dyspnea especially if the physician observes the patients take these deep breaths (overventilation) when they complain they cannot get enough air. It has often been helpful in my experience to find that, after an individual has complained bitterly of not being able to breathe even while at rest, the vital capacity of the lungs proved to be

normal. It is almost impossible for an individual to have distressing breathlessness from cardiac failure and yet have a normal vital capacity of the lungs. These considerations apply with equal force even when definite organic heart disease is present. Many patients with well-compensated valvular disease have dyspnea of neurogenic origin. The vital capacity of the lungs may be normal, the prognosis will be good and treatment must be directed at the functional state. Here also it is obvious that the proper interpretation of functional dyspnea will avoid errors in diagnosis, prognosis and treatment.

There is still another type of functional or neurogenic dyspnea that is different from that called "sighing breathing." It takes on a variety of forms. I once saw a young girl who appeared well, had very few complaints and merely showed a very faint systolic murmur. The respiratory rate, however, was about 55 although there was no dyspnea and the mother was not aware of the rapid rate of breathing. In another case an hysterical Negro had a respiratory rate of 120 with otherwise normal findings. He had very few complaints and could lie flat. During sleep, however, the rate of respiration was normal. In two other cases there was marked dyspnea and orthopnea with very noisy and rapid breathing. One case was first regarded as due to serious heart disease, then to bronchial asthma. The patient was cured by psychotherapy. The other was a most extraordinary case featured by terrific attacks of suffocation. The patient even had an exploratory operation on the mediastinum in one hospital and a tracheotomy in another. He also seemed to be cured, at least temporarily, by psychotherapy. These may be regarded as instances of hysterical dyspnea.

Consideration must also be given to the weight of the patient who complains of breathlessness. Mention has been made of the fact that dyspnea may be the only early manifestation of cardiac failure. It is also well known that obese individuals frequently suffer from organic cardiovascular disease, especially hypertension. What is not fully appreciated is that obesity itself in an otherwise healthy individual can produce shortness of breath. The stout person cannot breathe as freely as the lean. The diaphragm does not descend so readily and when the adiposity particularly involves the abdominal region the diaphragm is apt to be held in a high position and the vital capacity of the lungs is diminished. It follows, therefore, that not all obese patients who have dyspnea have heart failure, and that even when there is definite evidence of organic heart disease, the dyspnea may be only partly, if at all, the result of weakening of the circulation. There are many obese patients whom I have seen who had been treated for heart disease when I felt convinced they were not suffering from anything more than obesity. Such patients will do better on a dietary regimen judiciously followed, directed at a slow reduction in weight, than they will on digitalis therapy. In fact, even if the obesity is permitted to continue they do well, as this form of dyspnea is not progressive and is apt to be elicited only on effort, particularly climbing of stairs. It is evident that the converse of this is true, i.e., other things being equal, short-

ness of breath is more serious in a thin individual, for here despite the freedom of diaphragmatic movement there still is limitation in expansion of the lungs

Because breathlessness is the primary evidence of heart failure, whenever this is a major complaint either the patient or the doctor will quickly suspect the heart as the cause It must not be forgotten that there are other causes of shortness of breath Many diseases, particularly of the thoracic cavity, may produce dyspnea and occasionally they are overlooked when a weakened myocardium is thought to be present Some of these conditions are pneumonia, tumors of the lung miliary carcinomatosis of the lung, Hodgkin s disease, aortic aneurysm, pulmonary tuberculosis, bronchiectasis, pneumothorax and septicemia Under one set of circumstances or another the possibility of these diseases will need serious investigation, especially if the heart is found to be normal in size

In this connection mention should be made of deformity of the chest as a cause of true myocardial insufficiency The condition is called cardiorespiratory failure There are occasional instances when the thoracic cage is so deformed as a result of early infantile paralysis or tuberculosis of the spine, or because of congenital and developmental deformities such as 'trichterbrust or funnel chest,' that the position of the heart is markedly distorted Dilatation and hypertrophy of the cardiac chambers with congestive heart failure may result This is produced by mechanical factors such as kinking or constriction of the large vessels leading to or from the heart, or by changes in the lung It is now believed that the difficulty in respiratory function is much more important than the cardiac status Such cases progress like severe forms of chronic cor pulmonale from pulmonary emphysema, pulmonary artery disease or chronic pulmonary fibrosis Although this type of heart failure is quite rare it calls attention to the desirability of correcting as much as possible all chest deformities during the early years of life

The foregoing discussion amply justifies the point of view that the mechanism of dyspnea needs careful appraisal Various possible factors have different prognostic and therapeutic implications Only in this way will serious practical errors be avoided

17

The Clinical Significance of the Systolic Murmur

There has been a great deal of discussion concerning the causes and significance of a systolic murmur Much speculation has been expended on the physical and mechanical factors involved in the production of murmurs and considerable effort has been made to correlate the presence of systolic murmurs with anatomic diagnoses Despite this, much confusion remains, especially in the clinical interpretation of such findings Not so very long ago the detection of a systolic murmur meant heart disease and many an innocent, perfectly healthy person has been condemned as a chronic cardiac cripple, treated as such restricted in his activities and made to live his life in the constant fear that so commonly characterizes the life of an organic cardiac Others, as a result of such a mistaken diagnosis would develop the fullblown picture of cardiac neurosis and thereafter live with the handicaps that accompany this condition Some in defiance would lead a normal and active life for a great many years, to the amazement of the physician who originally made the diagnosis and who either outwardly or inwardly gave a grave prognosis

This point of view was quite prevalent before the First World War The presence of a systolic murmur was often regarded as meaning mitral regurgitation and this diagnosis carried with it the inference that the mitral valve was diseased During the war, however, there were so many young soldiers who had systolic murmurs and yet who were apparently well that much doubt was cast on its organic significance The result of this experience was that the pendulum gradually moved to the diametrically opposite position Whereas formerly all systolic murmurs were regarded as serious a teaching developed that systolic murmurs had no clinical significance whatever One authority went so far as to say ' throw the stethoscope away, ' emphasizing the importance of eliciting the early symptoms of heart failure Another expressed an extreme point of view that organic mitral insufficiency did not exist, that there was stenosis of the mitral valve or the valve was normal This latter opinion was based primarily on postmortem experience, in which it was found that when the diagnosis of mitral regurgitation was made the valve was either found to be normal or stenosed There is every reason, however, to believe that a middle course is much nearer the truth Systolic murmurs cannot be entirely disregarded nor do they always mean heart disease They

deserve our most careful consideration, for only in this way will those of importance be distinguished from the insignificant ones Furthermore, as will become evident in the following discussion, the proper interpretation of a systolic murmur may occasionally serve as the main clue to a diagnosis that otherwise will be entirely overlooked

It must be conceded that on examining most normal people no murmurs will be heard over the precordium Furthermore there is ample proof of the fact that disease of the mitral valve or a loss of the normal integrity of the valve leaflets can result in a systolic murmur If the mitral valve of a dog is cut there will immediately develop a systolic murmur that previously was not present The reverse of this, however, is not true for systolic murmurs can be present where the mitral valve is normal Apart from other factors to be considered later, a systolic murmur best heard at the apex of the heart can be due to regurgitation of blood through the mitral valve whether the valve is structurally normal or diseased If we have reason to believe that the valve is diseased the condition is regarded as "organic mitral insufficiency" If we believe that the valve is structurally normal the term used to designate the condition is "relative mitral insufficiency" This is a valid conception because there are numerous states in which for one reason or another the left ventricular cavity is enlarged or dilated and in this process of dilatation the mitral ring is sufficiently stretched so that normal leaflets can no longer completely close the orifice during ventricular systole and there results a regurgitation of blood There are, therefore, two conditions in which the mitral valve can be regarded as incompetent, one organic and the other functional

Organic mitral insufficiency generally results from a previous rheumatic infection Under these circumstances the valve is actually distorted, the free margins are apt to be thickened and retracted and although the slow process of stenosis has not as yet manifested itself, so that there still is free flow of blood during diastole from auricles to ventricles, there is a regurgitation through the incompetent valve during systole It is often difficult to be certain whether or not a true organic mitral insufficiency exists because there need be no symptoms referable to the circulation and no other evidence of cardiac disease not even hypertrophy of the heart However, when a patient has had a previous history of rheumatic fever or chorea, and there is an apical systolic murmur of greater than slight intensity, especially if there is some cardiac hypertrophy and an accentuated pulmonary second sound, it is a fair presumption that the mitral valve is organically diseased and insufficient It is no disproof of this contention to find a stenosis of the mitral valve on postmortem examination ten twenty or thirty years later When such a patient lives his span of life and dies of cardiac failure, mitral stenosis is very apt to be present Fatalities from heart failure occur but rarely during the stage of mitral insufficiency so that ordinarily we have no opportunity to confirm the diagnosis at the autopsy table There is one circumstance that does occur which enables us to see the mitral valve during the stage of organic insuffi-

ciency without stenosis, i e, subacute bacterial endocarditis In this way a fatal disease develops in a patient who has only a systolic murmur long before congestive heart failure would otherwise have occurred and we are permitted to examine a mitral valve that is diseased but not stenotic I have seen numerous such instances which have convinced me of the validity of making the diagnosis of organic mitral insufficiency

There is another type of structural mitral insufficiency that is very difficult to diagnose which may result from calcification of the annulus fibrosus. This can readily be seen on fluoroscopic examination, but produces no characteristic physical findings A moderate mitral systolic murmur is present in most but not in all cases It generally occurs in older persons and may be present without any other significant evidence of disease or disability, although it also is found associated with organic disease of the mitral valve itself This type of calcification of the mitral ring (in contrast to the leaflets) may or may not be associated with mitral regurgitation

On rare occasions organic mitral insufficiency without stenosis does lead to progressive cardiac enlargement even with marked dilatation of the left auricle and congestive failure It is not clear why most patients with simple mitral insufficiency do well for so many years and only a few develop heart failure rather rapidly Possibly the degree of valvular regurgitation, which is difficult to estimate during life and even postmortem, is the determining factor

Relative mitral insufficiency, on the other hand, is a common occurrence in a variety of conditions When the heart is enlarged and the left ventricle is dilated in patients with hypertension, syphilitic aortic insufficiency or myocardial disease from any cause, a systolic murmur of varying intensity is frequently heard at the apex There results a relative incompetency of the valve without any true progressive disease of its structure It is of some importance to bear in mind the distinction between organic and relative insufficiency, for in the former we fear the eventual development of mitral stenosis or the subsequent complication of bacterial endocarditis, while with the latter neither of these conditions will develop no matter how long the patient lives

Unfortunately many systolic murmurs cannot readily be classified in either of the two groups just mentioned It is this which has led to so much confusion and has given rise to such terms as accidental, cardiorespiratory, hemic or functional murmurs It is not the intention to explain or analyze all these murmurs but rather to present some points of view that have developed from an extensive clinical study of the systolic murmur and which have proved useful in a practical way

At the outset progress will be impeded if we do not start with clearly defined terms By definition a systolic murmur, no matter how faint, must have duration, it must last an appreciable interval into systole between the first and second heart sounds Because systolic murmurs have been so generally regarded as benign and inconsequential, medical students and young house officers have developed the habit of finding many

systolic murmurs that never exist They know that nothing will be said about it if some one else fails to hear it, especially as we know that faint murmurs may come and go, but they fear missing a murmur if it is present The result has been a looseness and carelessness in examination and in terminology Frequently I have failed to hear any murmur whatever on most careful auscultation when others have described a systolic murmur One additional reason is that with the definition just given, a prolonged or impure first heart sound that frequently is heard, especially in thin-chested individuals with hyperactive hearts, will not be called a murmur for there is no true bruit extending into systole With this definition in mind it is surprising how often classical instances of mitral stenosis will show no systolic murmur The characteristic diastolic or presystolic rumble will be heard ending with a snapping first heart sound but no murmur will be heard during systole

The other essential in this discussion is that we must indicate the intensity of the systolic murmur Quantitative descriptions have become useful in describing other findings, for example, the amount of albumin or sugar in the urine, the degree of jaundice or peripheral edema A very large trace of albumin for instance, is not customarily seen in the simple albuminurias accompanying fevers, while it is suggestive of nephrosis, on the other hand a slightest possible trace of albumin does occur with the former and not with the latter Similar notations are applicable in describing systolic murmurs For some years I have used the following terminology and although at first glance it may seem cumbersome, within a very short time those who have tried to use it have found it simple and reasonably accurate, so that different observers would coincide very closely in their decisions Systolic murmurs are divided into six gradations Grade I intensity is the faintest that is audible on the most careful auscultation Although faint it must have an appreciable duration This type of murmur is frequently overlooked if the examination is only casual It is generally not heard at all during the first few seconds of auscultation but then becomes audible In fact, if a faint murmur is heard immediately it generally means that it is grade II Grade VI intensity is the loudest murmur that one ever hears These murmurs are rare and are such that they can be heard with the stethoscope away from the chest wall The other four gradations (II to V) fall in between these two extremes They may be called "very slight," "slight" "moderate," "loud," "very loud" and "loudest possible" murmurs With a short period of practice different observers will find that they generally will use the same notation and almost never vary more than one gradation in intensity

With the foregoing as a background it was found that on examining over one thousand so called "normal" or noncardiac individuals, systolic murmurs of grade I intensity were fairly common, those called grade II were less frequent, and those of grade III quite rare The interesting point was that in every instance in which a systolic murmur of grade III intensity was heard, although such patients were in the hospital primarily for some condition unrelated to the heart, such as prostatic disease, hernia or

hemorrhoids, definite evidence of organic heart disease was found In fact, this was also true of many patients who had grade II murmurs There were several factors undoubtedly involved in the production of some of the murmurs of grade I and II intensity, and in interpreting such murmurs these factors need to be carefully considered These are fever, anemia, tachycardia, hypertension, hyperthyroidism and possibly nervous excitement The last of these probably produces its effects primarily through its increase in rate It does not follow that these conditions invariably produce systolic murmurs There are other influences involved that are at present poorly understood which determine whether a murmur will result or not However, it is fairly certain that they can be the specific cause of a murmur and therefore need to be carefully considered in estimating the significance of such murmurs, although they alone will not account for murmurs of grade III intensity or louder It is obvious that when the above six conditions are known not to exist, then the presence of a systolic murmur of more than grade I intensity is likely to be due to organic heart disease

There is one possible mechanism in the production of a systolic murmur, which is common to some of the conditions, that I wish to discuss This is the velocity of blood flow The rate at which the blood flows around in the circulation (not the heart rate) is increased in hyperthyroidism, anemia, fever and exercise An increase in the basal metabolic rate of the body will be accompanied by a speeding up of the velocity of blood flow It is interesting that many patients with these conditions also have systolic murmurs I have frequently observed grade I or II systolic murmurs in patients with hyperthyroidism and found that they disappeared after the basal metabolism was brought to normal by subtotal thyroidectomy Likewise, perfectly normal young men who show no murmur will almost invariably develop a grade I or II systolic murmur directly after a short brisk effort, such as running These murmurs will also disappear as the heart quiets down These murmurs have often been explained on the basis of temporary dilatation of the heart with relative insufficiency of one valve or relative stenosis of another X-ray examination of the heart in some of these conditions has failed to show any dilatation and in fact, at times, such as directly after a brisk effort, the heart seems to be a bit smaller than it is at rest May it not be that the murmur is produced because the blood is ejected with a snap? It has been shown that there is a speed with which a fluid running through a tube does so without producing eddies and bruits and beyond which such disturbances do arise This explanation seems logical when measurements of the velocity of blood flow show it to be accelerated However, even when this is normal, a systolic murmur can result from the same mechanism if we assume that the speed of ejection from the heart is accelerated for a short distance up through the aorta and pulmonary arteries, although the rate of flow through the entire circulation is unaltered In any case there seems to be some relationship between the rate of ejection of blood from the heart and the development of a systolic murmur

One by product of this study was the finding of a transient systolic murmur developing almost invariably in normal persons after a brisk effort. This is of some importance, for it has become the habit of physicians who examine for insurance companies or civil service commissions to make such an exercise test and to draw certain inferences from the appearance of a systolic murmur. Although such a test can be of great value in uncovering mitral diastolic or presystolic murmurs that otherwise might be entirely overlooked, it is obvious that no significance whatever can be attached to the appearance of a systolic murmur after effort.

When a deliberate and careful attempt is made to interpret the significance of a systolic murmur, eliminating the factors discussed in the foregoing paragraphs, we are still left with a considerable number that are difficult to explain. Let us suppose that the heart is slow, there is no fever, hypertension anemia or hyperthyroidism and a systolic murmur is present. If it is a grade III murmur, one is almost certain to find other evidence of organic disease such as cardiac enlargement, a systolic thrill, a diastolic murmur or significant electrocardiographic abnormalities. If it is of grade I intensity, unless other evidence of heart disease is present, no significance can be attached to it, although even some of these I believe eventually prove to be rheumatic or organic. If, however, the intensity of the murmur is grade II a most careful investigation must be made for a possible rheumatic background. Apart from the past history of rheumatic fever and chorea which will often be lacking, inquiry should be made of a family history of rheumatic fever or valvular disease and one should try to elicit a history of early nosebleeds, vomiting spells, undue nervousness or sweats or some unexplained illness in childhood. The point is that many rheumatic infections mask themselves in such obscure fashions and some do no more than produce a systolic murmur. In this way patients with grade II systolic murmurs will often be identified as having some form of rheumatic valvular disease. Others will be found to be suffering from some form of congenital heart disease such as patent ventricular septum, pulmonary stenosis, atrial septal defect, patent ductus arteriosus or coarctation of the aorta.

Having such indefinite evidence, it is not easy to convince oneself and much more difficult to convince others that a heart showing hardly anything more than a systolic murmur is not normal. This is particularly so because in some such cases, even those followed for a great many years, nothing further happens to incriminate the heart. This unfortunately does not occur in all cases. I have seen numerous instances in which patients with so called benign systolic murmurs later developed conditions which proved that the original murmur, although in no way impairing the efficiency of the circulation, was due to an inherent structural defect. In some a later bout of typical rheumatic fever serves to indicate that the early murmur probably was also rheumatic. In many the subsequent development of subacute bacterial endocarditis also proves that the original murmur was due to a minor rheumatic valvulitis or to some congenital defect because this complication is almost never superimposed on a pre-

viously normal heart In still others as years go on, a systolic murmur that was regarded as functional or insignificant becomes readily identified as due to aortic stenosis, when a systolic thrill develops over the upper or midportion of the precordium This has proved to be so in cases that I have followed even when the systolic murmur was originally more prominent at the apex than in the aortic area In some the eventual appearance of the signs of mitral stenosis indicates that the early murmur was due to a mitral valvulitis I have frequently followed patients who were well but showed a systolic murmur and found that ten years later a basal thrill and even x ray evidence of calcification of the aortic valve developed This is to be expected because long before a degree of stenosis of the valve has occurred sufficient to produce the classical signs, there must have been a time when the constriction was slight and the systolic murmur only faint

A few illustrative experiences will help to emphasize how the proper interpretation of an "insignificant systolic murmur" may lead to a correct diagnosis that would otherwise be overlooked It was my duty some years ago to make routine examination of forty members of the first year class of the Harvard Medical School There was only one who showed a systolic murmur This was of grade II intensity This young man felt well and did not consider himself sick There was no previous history of rheumatic infection, no hypertension and the heart showed no other abnormalities One might have dismissed this finding as of no importance and called it a benign or functional systolic murmur On closer scrutiny it was found that his skin was somewhat moist and hyperemic and that there was a very slight tremor of the fingers The heart sounds were also hyperactive Although there was no exophthalmos or thyroid enlargement, hyperthyroidism was suspected, for which he was later treated as the basal metabolic rate was found to be +45 per cent In this case the systolic murmur was the only feature that led to a diagnosis which otherwise would hardly have been suspected

In the following instance (referred to in Chap 10) attention to a simple systolic murmur also was the main clue to the diagnosis which had been entirely overlooked A day laborer was sent into the hospital with a diagnosis of stone in the left kidney A day or two before, he was taken with a sudden sharp pain in the left loin extending around the left part of the abdomen and down to the genitals There was gross hematuria which was noted by the patient himself He had been working when this occurred and complained of very little else Examination was essentially negative except for a grade II apical systolic murmur The urine showed gross and microscopic blood The temperature and pulse rate were normal The same diagnosis of left renal stone was made by the hospital staff, no attention being paid to the systolic murmur When the patient was shown at a staff conference, in my attempt to explain the presence of the murmur I elicited some additional information by direct questioning Although he had been working steadily he had not been feeling quite so well during the previous few weeks He had also known that he had some sort of a

murmur for years but paid no attention to it as it never troubled him. On the basis of this I ventured the diagnosis of subacute bacterial endocarditis with an embolus to the left kidney This suggestion was rather ill received but that afternoon, although the temperature was only 99 2° F, a blood culture was taken which was positive for *Streptococcus viridans* Cystoscopic examination which was contemplated was not performed and the progress was typical of subacute bacterial endocarditis Postmortem examination performed some months later showed typical vegetations engrafted on an old rheumatic mitral endocarditis This case also illustrates that during the years when the patient felt well and showed nothing more than a systolic murmur, the diagnosis of organic mitral insufficiency would have been justified

In this final case the interpretation of a systolic murmur was helpful in deciding upon the proper treatment A retired foreman, 60 years of age, came under my care because of shortness of breath, especially at night He was evidently suffering from congestive heart failure The heart was enlarged, the rhythm was regular and there was a grade II basal systolic murmur No thrills or diastolic murmur could be detected There was no hypertension or rheumatic or luetic past history and there was no history of angina pectoris The Wassermann reaction was negative The question arose here whether or not we were dealing with a case of syphilitic aortitis This deserved serious consideration for in about 15 to 25 per cent of such cases the Wassermann reaction is negative Although I do not believe that disease of the wall of the aorta (so called roughening) is a factor in the production of basal systolic murmurs, this has been the current teaching and the systolic murmur might be explained in this way X-ray examination however, showed definite calcified stenosis of the aortic valve This finding not only made the anatomic diagnosis but served to rule out lues as an etiologic factor for syphilis never produces stenosis of valves In this way the patient was spared a course of antiluetic treatment which not only would have been useless but might have been harmful When his clinical condition improved as a result of ordinary treatment for congestive heart failure a typical systolic thrill of aortic stenosis was felt and the murmur increased in intensity to grade III It is obvious from this and many similar experiences that a murmur may be faint when cardiac function is poor and louder as the circulation improves Furthermore, a faint murmur may have greater significance when heard in an obese individual or in a patient with an emphysematous chest than in a thin person The distance between the origin of the murmur and the external chest wall will necessarily affect the loudness of murmurs

Finally, there are some general remarks about systolic murmurs that are not out of place Too much emphasis has been placed on the transmission of murmurs in deciding whether they are functional or organic The louder the murmur, the greater will be its transmission, and very faint murmurs are not transmitted The location and the loudness are important, not the transmission A loud aortic systolic murmur is transmitted to the neck and a similar murmur in the mitral area to the axilla

because these locations are near the point of maximum intensity of these murmurs A very loud murmur no matter of what origin will be heard over a large area I question the importance of transmission of murmurs through the blood stream I have heard grade VI murmurs transmitted down the arms and audible over the olecranon process of the elbow when they could not be heard over the brachial artery They seemed to be transmitted well through bone Furthermore, the distinction between organic and relative or functional murmurs often has erroneous connotations The former sounds more serious than the latter, whereas relative dilatation of cardiac cavities resulting in functional systolic murmurs is apt to signify a graver condition of the heart muscle There are some nervous persons who show hyperactive hearts and a systolic murmur without other evidence of organic disease who fall into a group of potential hypertensives They often have a very slight fever, show flushing of the skin of the neck, and as they are followed for years they eventually develop permanent essential hypertension

It must not be inferred from the foregoing discussion that all systolic murmurs should be regarded with gravity For the most part we should do as we have been doing in the past Patients with systolic murmurs and no other evidences of circulatory embarrassment may remain in good health for many years or even indefinitely If there are no symptoms of cardiac weakness they should be allowed to do as they please There really are no restrictions that they need They may be allowed to enjoy the kind of physical activities and sports that produce no ill effects This advice is proper not because it is felt in all cases that the systolic murmur has no meaning, but rather because there is no advantage in enforcing rest or denying such individuals the ordinary pleasures of life However, it must be clear that an attempt should be made to ascertain the cause of the murmur As an aid in this direction proper terminology and estimation of the intensity of murmurs should be employed In this way diagnosis will become more accurate, treatment in some cases will be directed more intelligently and vague terminology will become more clarified

18

The Patient with Heart Disease As a Surgical or Obstetric Risk

The physician is frequently asked by his surgical colleague whether a certain patient can stand an operation This is by no means as simple a problem as it seems for it requires an answer to three different questions First the physician must help in the diagnosis of the condition and decide whether the problem is surgical after all As will be seen there are many cardiac affections that present features which make them similar to and confused with acute surgical abdominal emergencies Secondly, when there is an obvious disease that is amenable to surgical treatment one has to decide whether or not the prognosis of the cardiac condition is good enough to warrant subjecting that patient to a major operation Finally, when an operation is contemplated the physician should have some idea as to the surgical or operative mortality in various cardiac disorders

With regard to the first problem a physician often can spare a patient an unnecessary operation by making a correct diagnosis when the condition was supposed to be an acute surgical abdomen In children, pericarditis or acute rheumatic fever may be accompanied by abdominal pain and tenderness, slight fever and leukocytosis and even nausea and vomiting When joint pains are absent, acute appendicitis may be closely simulated Inasmuch as there is no specific test for either condition, at times it will be necessary to perform an abdominal exploration to be sure of the diagnosis, for it is safer to find an occasional normal appendix than to overlook appendicitis that develops into a fatal peritonitis There will be occasions, however, in which the detection of some of the subsidiary evidences of rheumatic infection, such as epistaxis, a positive family or past history of rheumatic fever or rheumatic heart disease, will be sufficient to make one doubt the diagnosis of acute appendicitis and delay operation After such a delay of twelve to twenty four hours it may then become quite clear that there is nothing surgical about the condition

On very rare occasions the sudden development of auricular fibrillation, in one suffering from some form of organic heart disease, may be attended by acute pain and tenderness in the epigastrium or right upper quadrant, nausea, vomiting, abdominal rigidity, slight fever, leukocytosis and slight icterus The whole picture may resemble an acute cholecystitis I have

seen such a patient upon whom a cholecystectomy was performed following which there was satisfactory recovery About one year later I saw the same patient go through a similar spell and she recovered on digitalis therapy Reviewing the data of her previous experience it was clear that she had mitral stenosis and developed a paroxysm of auricular fibrillation which resulted in abdominal symptoms presumably due to an acutely engorged liver After the gallbladder, which contained no stones, was removed the auricular fibrillation spontaneously disappeared and recovery was satisfactory The second attack was like the first and was again accompanied by a sudden paroxysm of a rapid irregular heart action with an enlarged tender liver This time the symptoms all subsided on appropriate treatment for the cardiac condition It is obvious that the previous operation was unnecessary

Another circulatory condition that produces symptoms resembling an acute surgical abdomen is embolism to one of the abdominal viscera An embolus to the spleen or the kidney can result in sudden pain and tenderness in the abdomen with fever and leukocytosis This is apt to occur in those cardiac patients who show evidence of mitral stenosis, auricular fibrillation or subacute bacterial endocarditis The treatment is expectant and supportive and not surgical When such an embolus involves the mesenteric vessels surgical treatment may be necessary The diagnosis, however, is by no means simple and fortunately such a complication requiring operative resection is rare

Finally, acute coronary thrombosis may resemble very closely an acute surgical condition of the upper abdomen This has been adequately discussed in a previous chapter (Chap 6) Suffice it in this connection to recall that all available methods may be necessary to make this differential diagnosis and to avoid the error of operating on a patient who is in the throes of a desperate cardiac affliction The resemblance may be to acute gallstone colic, perforated peptic ulcer or acute pancreatitis An electrocardiographic study may be invaluable and distinctive under these circumstances Physicians have become aware of this diagnostic difficulty and now must be alert not to make the opposite error and overlook an abdominal disorder requiring immediate surgical intervention

There are other acute circulatory emergencies that bring up the question of some surgical intervention in which the physician s judgment may aid in determining the proper treatment At present, for example, it is a matter of careful judgment whether or not to try to remove a peripheral embolus to an extremity Embolectomy is only of value the first six to twelve hours and it must be remembered that gangrene rarely develops in the arms The use of suction apparatus and anticoagulant therapy seems to offer a fairly satisfactory method of treatment Suction apparatus is really a peripheral pump and produces an alternating state of pressure and suction to the affected limb and thereby improves the blood flow through the part involved Furthermore, novocain injection of the sympathetic nerves to the limb involved may help to relieve the additional spasm that takes place with embolism, and freezing of the extremities is

now being employed. It is clear, therefore, from the preceding discussion that when a physician is asked whether a patient can stand an operation, he should first try to decide whether there is any surgical problem involved at all or whether the cardiac disorder itself and its complication may adequately explain the difficulty.

The second point to determine is whether the life expectancy of the particular patient suffering from heart disease warrants undertaking the surgical procedure that is contemplated. Is it to be expected that the patient will live long enough to enjoy the results of the operation to make the temporary discomfort and risk worthwhile? This involves an estimation of the prognosis in the type of cardiac disease that is present, frequently a difficult matter. However, it is often fairly obvious that the patient cannot be expected to live more than a year or two and then one would hesitate in recommending an operation that is not urgent or for a condition that can be treated, even if less satisfactorily, by nonsurgical methods. Too frequently women with hypertensive heart disease or mitral stenosis are subjected to pelvic operations, only to succumb to circulatory failure within a year or so after the operation. For many who survive the operations there must be some who make up the ordinary surgical mortality that attends such an operation. Patients with fibroid tumors of the uterus who also suffer from advanced heart disease with an accompanying poor life expectancy are much better treated by radiation than by surgery. Similarly, operations such as that for prolapse of the uterus or pelvic repair might well be avoided in such patients. Instead, nonsurgical methods may be employed. The same may be said for a simple hernia. In a word, whenever possible it is wise to employ simple nonsurgical methods of treatment in patients with advanced heart disease although one should not hesitate to subject them to operations that are more urgent such as that for acute appendicitis.

The third consideration is an attempt to estimate the surgical mortality in patients suffering from different types of organic heart disease. Some years ago I made a review of 414 cases subjected to 494 operations. The surgical problems involved could all be regarded as major and the cardiac abnormalities were all organic. In order to determine the role played by the heart in the outcome, deaths were divided into two types, 'unexpected' and 'inevitable.' In the former group were included all those cases in which it seemed that the patient would not have died had no operation been performed. This, therefore, included all unexpected circulatory disasters such as coronary thrombosis, cerebral hemorrhage, embolism or circulatory failure. Even the ordinary complications frequently seen in noncardiac cases, such as postoperative pneumonia, were regarded as 'unexpected.' Among the 'inevitable' deaths were those due to the underlying disorder of the heart at about the same time as they might have died irrespective of the operation. This can be illustrated by the instance in which death occurred forty-eight hours after amputation of a gangrenous leg in a patient who was moribund from an acute coronary thrombosis with a femoral embolus. These 'inevitable' deaths are of no

importance in this discussion as they do not reflect the added risk that heart disease produces in withstanding operations. In this way it was found that the total operative mortality was 12.1 per cent and the "unexpected" mortality was only 6.3 per cent. This latter figure readily indicates that as a group patients with heart disease undergo surgical procedures fairly satisfactorily.

A more detailed analysis of the various types of heart disease disclosed some interesting relationships. There were only three "unexpected" deaths in 147 operations performed on patients with valvular disease, i.e., a mortality of 2.1 per cent. In 167 operations on patients with nonvalvular heart disease (hypertension, chronic myocarditis, etc.) there were eight "unexpected" deaths or a mortality of 4.9 per cent. Curiously enough the mortality among patients with permanent auricular fibrillation was also very low as there were only three "unexpected" deaths in 108 operations (3 per cent). Disease of the coronary arteries was found to increase the risk appreciably. In forty-one operations on patients with angina pectoris there were three deaths (7.7 per cent) and in twenty on patients with coronary thrombosis there were eight "unexpected" deaths (40 per cent). This latter figure was unduly high for it included some patients who were operated on in the midst of an acute coronary thrombosis either by mistake or when such an operation could readily have been delayed. There was one death in thirteen instances of syphilitic aortitis and none in six patients with paroxysmal tachycardia, three of whom had attacks during the operation. Congestive heart failure was found to add considerably to the operative risk as there were seven "unexpected" deaths in fifty cases (14 per cent). Dividing the patients into those with and those without nephritis showed that the mortality of the former was 14.8 per cent whereas in the latter it was only 4.9 per cent. Hypertension, on the other hand, was found to produce very little effect on the mortality. Patients with systolic readings over and under 160 mm. had mortality figures of 7.3 per cent and 5.9 per cent respectively.

It is clear that patients with organic heart disease who are well compensated in general stand major operations satisfactorily. The risk increases if there is congestive heart failure and although at times it is necessary and advisable to operate in the presence of congestion, whenever it is possible to delay until a better state of compensation can be established the operation should be postponed. The additional presence of nephritis adds to the surgical risk. The presence of angina pectoris likewise carries a somewhat greater hazard primarily because such persons are always subject to sudden coronary thrombosis or sudden death. The risk in cases with coronary thrombosis will not be so great as was indicated in this analysis if an accurate diagnosis of the cardiac condition is made and if operations are postponed until a sufficient time has elapsed after the attack of coronary thrombosis.

It is difficult to study the effect of different anesthetics in relation to surgical mortality in patients with heart disease. Individual clinics develop their own peculiar customs and preferences. In some ether is used a

great deal, in others spinal or local anesthesia is favored and in others ethylene is a common choice. In the group discussed above various methods were employed (ether, local and intraspinal). There does not seem to be sufficient comparative data with regard to the relative merits of the various anesthetics to enable one to draw any conclusions. The choice must remain for the present an individual matter and rest upon the combined decision of the physician, the surgeon and the anesthetist. In general it may be said that when a local anesthetic will not suffice, ether is tolerated very well by patients with heart disease.

Finally a word must be said concerning the cause of death in these patients. They are naturally subject to the same hazards as a patient having a normal heart. Postoperative pulmonary complications are still the most common cause of surgical mortalities. Sepsis, phlebitis and hemorrhage will inevitably occur at times. The added difficulties that come because heart disease is present are the so-called "accidents" of heart disease. It seems that as a result of the operation, in some cases, emboli may become dislodged from silent mural auricular thrombi producing pulmonary infarction, hemiplegia or other arterial occlusions. Likewise occasionally an attack of coronary thrombosis may be precipitated by an operation. In this regard a marked fall in blood pressure in any patient having coronary artery disease must be avoided if possible because of the danger of precipitating coronary thrombosis. At times when an operation is performed for some septic process or when an infection results from an operation, subacute bacterial endocarditis may develop as a direct result of this infection. This can now be prevented by penicillin therapy. The ordinary type of congestive heart failure is only rarely precipitated by the operation unless infection or one of the above "accidents" has occurred. The added load or work which the heart is asked to perform as a result of the operation itself is no greater than the patient has already been demanding of that heart before the operation. As far as heart strength is concerned anyone who has been able to walk moderately without much discomfort is subjected to no greater hardship in undergoing an operation. The "unexpected accidents" of heart disease, however, cannot be accurately predicted and they constitute the main factor in increasing the risk from surgery.

HEART DISEASE AND OBSTETRICS

There is some similarity between the problems involved when a patient with heart disease becomes pregnant and when one is to undergo a surgical operation. There is one important difference, however, in that the former condition is to a great extent predictable and voluntary. This increases the responsibility of the physician for he will be asked whether pregnancy should be contemplated or after it has occurred whether it should continue. The intelligent answer to these questions will require not only a knowledge of diagnosis and prognosis of heart disease but also an insight into the social and economic life of the family involved.

There are several cardiac conditions that can readily be dismissed as

having no influence on the question of pregnancy, for the response of the heart under these circumstances is practically the same as when the heart is perfectly normal These conditions are all the forms of functional heart disease and well-compensated mitral insufficiency Patients with benign irregularities of the heart or those showing insignificant faint systolic murmurs may be regarded as taking a normal risk The same applies to those who have a louder mitral systolic murmur with or without slight cardiac hypertrophy in whom a past history of rheumatic fever or some other features indicate that there is an organic mitral regurgitation Provided there has been no evidence of congestive failure and the patient's response to exercise is satisfactory, such patients can be regarded practically as undergoing a normal risk in pregnancy

On the other hand there are some cardiac conditions which when present make pregnancy highly inadvisable If a patient is suffering from subacute bacterial endocarditis it is obvious that the seriousness of the underlying disease does not warrant undertaking or continuing with pregnancy unless the infection responds to chemotherapy If the mother is suffering from active rheumatic fever it is undesirable that she be pregnant at that time Apart from any ill-effects that the pregnancy may have on the mother, and it is not certain that there are such ill-effects, the child may be born with a rheumatic carditis, for there are instances of acquired intra uterine rheumatic heart disease

The main problem arises in patients with mitral stenosis and aortic valvular disease Of first importance is the state of compensation of the circulation If there is any evidence of congestive heart failure or if such failure has once been present in the past, it is best to advise that no further pregnancies be undertaken Even when an apparently satisfactory state of compensation can be established by appropriate medical treatment, the risk of recurrent heart failure is too great and the life expectancy of the mother is too short to make it advisable for such a woman to go through pregnancy If it is undertaken, a high maternal mortality must be expected In general, women with rheumatic heart disease with congestive failure or with auricular fibrillation will have a maternal mortality of 30 to 50 per cent and only 50 per cent will have viable children On the other hand, if there is no objective evidence of heart failure or dypsnea is either entirely absent or only of a slight degree women with mitral stenosis, aortic stenosis or aortic insufficiency should be permitted to go through pregnancy The only exception to this is the presence of permanent auricular fibrillation This latter condition is generally associated with some signs of heart failure and for that reason alone will contraindicate pregnancy, but even when such has not been the case patients with auricular fibrillation are generally suffering from a disordered circulation that is too advanced to warrant taking the risk Women manifesting those conditions that contraindicate pregnancy, i e, permanent auricular fibrillation, present or past congestive heart failure, should, therefore, be cautioned to avoid pregnancy and should be instructed in the methods of contraception

There are differences in the advice which the physician should give

when the question of the first or of subsequent pregnancies arises What has been said above concerning well compensated cases of aortic or mitral disease applies primarily to those women who contemplate their first pregnancy Even when there is some doubt as to the exact state of the heart a slight added risk might be hazarded for the joy of having a child in contrast to a childless life The situation is not quite the same if the patient already has one or more healthy children Realizing that there is always a greater risk among pregnant organic cardiac patients than among normal women, no matter how apparently trivial the disease may be, I feel that if there are already three children, under no circumstances should any more pregnancies be undertaken It is not pertinent to the question to recall the instances in which women with mitral stenosis have satisfactorily borne eight or ten children We see as patients only those who have survived Many of the other multipara have succumbed, leaving their children motherless

A distinction is made when there are already three children because that constitutes a satisfactory family both from a social and an individual point of view It must be remembered that there is a strong familial factor in rheumatic heart disease and that with numerous pregnancies there is a great likelihood that one or more of the children will eventually suffer from rheumatic heart disease If precautions are taken in families with stigmata of important hereditary nervous disorders like insanity, why should not similar considerations be given to this disabling form of heart disease? Furthermore with numerous pregnancies among people of humble or modest means, the task is not ended with recovery from the confinement The rearing of several infants and children without the aid of nurses and maids may prove to be a greater task than the pregnancy itself and more than the disabled heart of the mother can stand The social and economic status of the patient, therefore deserves careful consideration

When there is already one child and further pregnancies are contemplated the physician would do well to encourage a second pregnancy Both the first and those subsequent pregnancies that are planned should take place during the early years of married life Some women either through fear or improper medical advice delay this decision and lose valuable years thereby Unless some acute episode has taken place, the cardiac condition does not generally improve with advancing years so that a pregnancy will be better borne earlier rather than later Furthermore, the mother will enjoy the life of the children longer if they are borne during the early years of married life A second pregnancy should be encouraged if there are no contraindications because a family is not secure with one child Through some unfortunate accident or illness that child may die Two healthy children therefore, consolidate the family and also prevent the difficulties that often arise from a spoiled single child When it comes to the third pregnancy one may rightly exercise a choice In a word I urge the first pregnancy, advise the second and consent to the third

The foregoing considerations apply to those women who are not preg

nant and come to the physician for advice The problem is somewhat different if the patient is already pregnant At the outset it must be emphasized that every woman with organic heart disease should be cautioned to consult her physician at the first suspicion or indication of pregnancy, so that a decision may be made early during pregnancy This is particularly important in public cardiac clinics when patients are seen only at infrequent intervals If a menstrual period is missed it should quickly be determined whether pregnancy has occurred This can be done quite satisfactorily by means of the Aschheim Zondek or other pregnancy tests Once it is known that the patient is pregnant a decision should be made whether or not abortion should be advised If there is or has been definite congestive heart failure or if permanent auricular fibrillation is present, termination of pregnancy is indicated The procedure involved is comparatively harmless during the first few months and, therefore, the deci sion should be made early If it is thought advisable that abortion should not be induced the patient should be advised concerning her activities and should be under frequent medical observation It rarely is necessary in these cases to administer digitalis unless there is congestive failure or permanent auricular fibrillation Women should be cautioned against overdoing Frequent shopping trips may precipitate symptoms of congestive heart failure There still prevails in the minds of many women and some physicians that an expectant mother must 'harden herself by deliberate the trial of labor I have seen such a patient systematically iles a day over hilly ground at the advice of her obstetrician, month until she developed heart failure A respiratory infec a frequent cause of upsetting the state of compensation and as possible it should be avoided When a minor respiratory infec- has occurred more than the usual care and rest in bed should be dvise

evidence of weakening of the circulation will be a slight dyspnea a slight nonproductive cough and the presence s at the bases of the lungs Inasmuch as some subjective y have been present before the pregnancy occurred, and many n without heart disease may complain of slight dyspnea during ancy, it is no simple matter to appraise this symptom It has proved very helpful to me in following these patients to watch the vital capacity of the lungs When the patient progresses favorably it will be found that the vital capacity of the lungs, which is an excellent objective index of the degree of breathlessness, will remain essentially unchanged throughout pregnancy It is surprising that even at the eighth month when the abdomen is markedly distended by the enlarged uterus the movements of the diaphragm are not impeded sufficiently to lower the vital capacity By this simple determination one can obtain a check on the patient s clinical condition Obviously the physician should carefully examine the patient during these frequent visits, watching particularly for basal rales apart from observing the blood pressure and urinary findings Pitting edema of the legs which is such an important sign of congestive heart

failure is not trustworthy during pregnancies because many women manifest this as a result of pressure of the uterus on the pelvic veins or for other reasons

If any definite evidence of congestive failure is detected the patient should be kept in bed and treated appropriately If this occurs during the first three months and there is already one healthy child, abortion should be advised after the state of compensation has been improved, even if what appears to be satisfactory recovery of the heart has taken place If heart failure occurs in the latter part of pregnancy, the decision is difficult One needs to consider whether the child is viable or whether the pregnancy can be carried along far enough to obtain a viable child In some cases abortion will have to be carried out at the fifth, sixth or seventh month with the hope of saving the mother It is readily seen that there will be occasions when decisions are difficult and when the outlook is not very promising whatever course is taken However, once definite evidence of congestive failure has developed, that patient should be confined practically to strict rest for the remainder of the pregnancy

There is one type of heart failure that occasionally develops rather suddenly in pregnant women even when the heart was normal before pregnancy This may take the form of acute pulmonary edema during a toxemia of pregnancy with a rising blood pressure The mechanism is an acute left ventricular failure This condition is amenable to cardiac therapy Complete recovery can take place following the use of morphine, digitalis and phlebotomy when necessary In such cases the heart may return to normal and pregnancy may continue in the usual manner

There are sharp differences of opinion concerning the choice of operative procedure in pregnant women with heart disease Some obstetricians believe that a cesarean operation causes the least disturbance to a damaged heart Others maintain that it is best to permit the patient to give birth to the child in the normal way There probably is no single rule that will be applicable in all cases If it is expected that the labor will be short and easy and the state of cardiac compensation has been satisfactory, I believe a cesarean section should be avoided When, however, it has already been decided to perform sterilization, a cesarean operation should be done and the tubes should be tied off at the same time One should hesitate to do this at the time of the first pregnancy, as during the operation it is not known whether the child is viable or in normal health I recall an instance in which sterilization was performed at the time of the first confinement and the child died during the first twenty four hours The mother recovered satisfactorily and remained well-compensated She probably would have been able to go through one or two more pregnancies, but of course the sterilization prevented conception Sterilization should, therefore, be advised when it is certain that under no circumstances will the mother be allowed to go through any further pregnancies In my own experience it has seemed that pregnant cardiac women do very well with ether anesthesia and tolerate an abdominal section with very little difficulty When heart disease is advanced and the state of the

circulation is somewhat in doubt this method disturbs them less than a delivery from below. The intelligent cooperation between the obstetrician and the cardiologist has brought about a tremendous fall in the maternal mortality of those suffering from heart disease. This has been well exemplified in the special cardiac clinics established in lying in hospitals, such as that instituted by Hamilton in Boston. We now are able to advise patients whether or not to undertake the hazards of pregnancy and then to guide them along to a successful termination with a surprisingly small risk.

When the above general rules are applied the maternal mortality will be less than 3 per cent. This is still twenty times as great as in normal women. There will be unexpected instances of congestive failure in some cases of mitral stenosis, or fatalities may occur from gross emboli. I suspect that the risk can be a good deal less than the mentioned 3 per cent if meticulous care is taken, and now that we have drugs like penicillin. In my own experience there has been no mortality whatever except among a few patients who became pregnant against my advice.

Finally, one may ask whether the life span of cardiac patients who recover is shortened by pregnancy. This is a difficult question to answer. Some statistics indicate that women with rheumatic heart disease (not dying in pregnancy) are five years younger at the time of death than women with comparable conditions who have not been pregnant. This difference may be due to the fact that some develop congestive failure, recover partly and succumb a short time after delivery. If heart failure does not develop it seems unlikely that life has geen appreciably shortened.

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Factors Concerning Prognosis in Heart Disease

All physicians at one time or another have had the disturbing experience of making an absolutely wrong prognosis while treating patients with heart disease This occurs even when the diagnosis is correct Such wrong prognoses are made both when the condition seems hopeless and the patient recovers, and also when the patient has been doing satisfactorily and dies unexpectedly It is particularly humiliating to the individual physician and harmful to the general medical profession when a hopeless prognosis is given and the attending physician is dismissed, if one of the irregular practitioners is called in and, in the natural course of events, the patient recovers Such an experience makes an everlasting impression on the parties involved and on their friends and serves to place in disrepute the entire medical profession It is not surprising that the lay public should feel as they do under these circumstances Mistakes in prognosis, more so than mistakes in treatment, are responsible for the growing activities and importance of the numerous unorthodox medical cults that prevail today The purpose of this discussion is to throw some light on the factors in the prognosis of heart disease so that such errors may be minimized

There are two parts to the problem of prognosis that frequently arise in cardiac patients The first concerns itself with the immediate and the second with the ultimate outcome The factors that concern the latter are very involved and difficult to measure It is almost futile to speculate as to exactly how long a patient with compensated aortic or mitral valvular disease will live There are so many uncontrolled and unpredictable influences at work that although one can formulate some average figures that roughly guide our concepts of life expectancy it is difficult to apply them to individual cases Two patients start out at the age of 15 years with apparently similar lesions, e g , a slight aortic or mitral insufficiency One carries on in good health to the age of 50 or 60 and the other picks up a minor infection that initiates subacute bacterial endocarditis at the age of 30 This formerly proved fatal The same marked differences in ultimate outcome characterize disease of the coronary arteries One patient with angina pectoris has a sudden exitus shortly after the first symptoms develop and another continues in fairly good health for a great many years When we realize that there may be such extreme variation in life expectancy among apparently similar cases, for the present it seems idle

to try to predict exactly how long many of these patients will live Perhaps it is just as well that prognosis is no more accurate It enables both the physician and the patient to be hopeful and not to feel that the day of judgment is absolutely fixed

The difference between predicting immediate and ultimate results is well illustrated by the influence of the presence of auricular fibrillation with valvular disease Whereas auricular fibrillation indicates a more advanced state of rheumatic heart disease and on the average the life expectancy is shorter in the rheumatic cardiac patients with auricular fibrillation than in those with regular rhythm, the immediate prognosis and expectations for improvement are better if this irregularity is found than when the rhythm is normal and the same degree of cardiac failure is present

It is obvious that in any form of heart disease, other things being equal, the more ill the patient seems to be, the more dyspnea, the more peripheral congestion and similar symptoms, the more serious will be the outlook On the other hand, there are many patients with marked symptoms and physical signs of circulatory embarrassment in whom the prognosis is a good deal better than in others with much milder evidences of the same type This occurs because other less obvious factors are not the same in the two groups of cases We must try to explain why some patients with heart disease unexpectedly do better and others do worse than predicted, and even why, in some cases that are considered entirely hopeless, recovery takes place

One simple aid in estimating prognosis is the heart rate at which the patient develops congestive failure If two cases of mitral stenosis and auricular fibrillation present the same degree of cardiac failure, but one has a heart rate of 130 and the other a rate of 80, in general it may be said that the prognosis is better in the one with the rapid rate Here it is evident that digitalis and other treatment may be expected to slow the rapid rate and effect a good deal more improvement than when the rate is already slow In other words, when the heart rate is slow and there is considerable peripheral edema, not so much improvement can be expected as when the rate is rapid This is especially true when the disturbance is one in which it may be predicted that slowing will be obtained such as is the case in auricular fibrillation

The size of the heart is of some importance in judging prognosis The larger the heart, other things being equal, the poorer the prognosis This does not mean that one patient with a large heart may not do better than another of a different type with a small heart I recall seeing a patient with mitral stenosis and auricular fibrillation who had marked congestive heart failure She improved strikingly on ordinary treatment so that all evidence of congestion disappeared, and in fact the vital capacity of the lungs, which had been very low on admission, rose to a normal level before leaving the hospital She walked up two flights of stairs with one of the physicians and showed less respiratory distress than he did as a result of this effort One might have predicted that she would do extremely

well Her heart, however, was very large, almost filling the chest, and she died within a year Such experiences are not uncommon

One of the most important factors that needs proper appraisal is the role that infection plays in producing symptoms and signs of heart disease It is evident that infection, although violent, may come to an end A patient may be desperately ill with respiratory distress, very rapid heart, even showing signs of generalized congestive heart failure, and yet recover so that all evidence of circulatory embarrassment disappears Such a patient may not only feel well and remain well indefinitely, but physical signs of heart involvement, such as murmurs, may disappear entirely This sort of dramatic improvement is not altogether uncommon in young people The reason that this can occur is because under such circumstances almost the entire picture that develops is due to an infection, generally rheumatic fever This is apt to be accompanied by a pancarditis with involvement of the pericardium, myocardium and endocardium This sort of an infection may not only terminate, but recovery from it can be practically complete I have seen such patients who were ill enough to be on the danger lists in hospitals but who recovered entirely and showed no signs or symptoms of heart disease in subsequent years If on the other hand, the same degree of circulatory failure occurred without an infection merely as an end result of a burnt-out, long standing chronic cardiac condition, recovery of the type described would be extremely rare Improvement may occur so that the patient may become ambulatory, but there will always remain a varying degree of disability which is generally considerable The inference to be drawn is that when infection of the heart is present, although the condition may be very critical and frequently fatal, one has reason to be hopeful for a satisfactory recovery is always possible

The role of bronchial dyspnea in heart disease needs special consideration in judging prognosis There are a great many people with bronchitis of the asthmatic type and emphysema There are also a great many individuals who have one form or another of heart disease It would be quite natural, therefore to find a certain number who happen to have both conditions Furthermore, dyspnea on effort or at rest and paroxysmal dyspnea are symptoms that are common to both heart disease and asthma It, therefore, becomes very important at times to determine how much of the respiratory distress is circulatory and how much is bronchial in origin The importance of the differentiation lies in the fact that the prognosis in a patient with marked dyspnea of bronchial origin may be excellent whereas if the dyspnea were mainly cardiac, the outlook would be quite grave There are numerous instances of mistaken prognoses because of the improper interpretation of the mechanism of dyspnea For cases illustrating this problem and for the details that help in avoiding such errors see Chapter 16

There are other influences at work in the production of dyspnea that have no direct bearing on heart disease which one must clearly distin-

guish, because in so far as they are present the prognosis is so much better Obesity in itself can produce shortness of breath when the heart is normal and, therefore, can be responsible for a part of the dyspnea when there is heart disease The same degree of dyspnea will have less significance in an obese than in a thin individual if the other findings are the same In fact, there are many obese patients in whom a diagnosis of heart disease is made because they complain of shortness of breath, yet who really have no structural disease of the heart Likewise, patients with nervous weakness and debility complain of shortness of breath and palpitation and often are regarded as having organic heart disease when the entire problem is functional Furthermore, there are many patients with compensated organic heart disease whose symptoms are in the main functional or neurotic in nature In so far as that is true the prognosis is so much better There is one particular aspect of this question that deserves attention I refer to a group of comparatively young individuals who complain of shortness of breath even while at rest They are apt to say that they cannot get enough air and will frequently be seen to take deep breaths (sighing breathing) They actually overventilate their lungs and still want more air, in contrast to patients with cardiac asthma who really cannot take a deep breath and who have true respiratory distress They will show no signs of cardiac failure and no evidence of serious organic disease and yet feel short of breath without effort When spontaneous paroxysmal dyspnea is of importance it will generally be accompanied by obvious signs of grave heart disease, whereas in this type of ' sighing breathing, examination will reveal no essential abnormality The distinction between these two types of dyspnea occurring when the subjects are at rest is most important for on the one hand the prognosis is excellent and on the other it is very grave

It is obvious that the facility with which the symptoms of heart failure (dyspnea, edema and chest pain) develop and with which they disappear under treatment is of some help in judging prognosis Edema of the legs that develops only at the end of the day and disappears overnight is less serious than edema that persists all day The same is thought to be true of anginal pain coming in some patients only if they hurry up a hill and in others even when they try to shave themselves Similarly, if the patient improves quickly, one may regard the outlook as better than if it takes many weeks to accomplish the same result Although there are exceptions to the foregoing principles, in general they will be found to be true

In following a case of mitral stenosis I have found that the development of hypertension is indicative of a better prognosis than one might otherwise expect As a group, the majority of patients with mitral stenosis will have died before they reach the age of 50 More recently we have learned that an appreciable number will live to 60 and some to more than 70 It is curious that hypertension is much more common in this older group of patients with mitral stenosis than in the general population for corresponding ages, despite the fact that mitral stenosis of itself in younger people is accompanied by a blood pressure somewhat lower than

the average Why this should be so is not altogether clear, although it may be that the original rheumatic infection that produced the mitral stenosis also had some insidious effect on the blood vessels causing the hypertension In any event, I have frequently been aided in giving a better prognosis than would otherwise be warranted by finding the blood pressure elevated For example the presence of a systolic blood pressure of 140 to 160 during some acute cardiac emergency like an infarct of the lungs with generalized pulmonary edema in a patient with mitral stenosis would lead one to hope for recovery although the patient may appear desperately ill Similarly if the blood pressure gradually rises to 160 systolic or more from year to year, a patient with mitral stenosis may progress more favorably than one would otherwise expect The development of moderate hypertension, therefore is a favorable sign during the course of mitral stenosis

It is well to contrast the prognosis of dyspnea and congestive heart failure in cases of mitral disease with that in aortic valvular disease Many patients have well marked heart failure with mitral stenosis, re cover compensation and again become ambulatory They may carry on for many years, going through repeated breaks in compensation On the other hand, when a patient with aortic stenosis or insufficiency, rheumatic or luetic once has dyspnea and peripheral edema his months are often numbered However, the aortic patient is apt to be stronger and suffer less from dyspnea than the patient with mitral stenosis until this final break in compensation does occur The effect of tricuspid stenosis on prognosis was discussed in Chapter 4 It is of interest that when this lesion is present the length of life is much greater after heart failure develops than in any other form of valvular disease

Of special significance in considering the question of shortness of breath is nocturnal paroxysmal dyspnea This occurs most frequently in patients with hypertensive luetic or coronary artery heart disease This symptom even more so than pulsus alternans gallop rhythm and bundle branch block carries with it a very poor prognosis Most patients will not survive the first development of nocturnal dyspnea more than two years There are numerous patients however whom I have seen with pulsus alternans and gallop rhythm who did well for more than five years We must try to individualize in our prognosis and it will be found that if some acute episode, which can disappear brought to light these grave signs they need not have the same hopeless outlook Many patients will show Cheyne Stokes breathing pulsus alternans and gallop rhythm during the height of a coronary thrombosis or an attack of paroxysmal tachycardia and yet recover and do well for years In fact, in some of these cases of paroxysmal tachycardia the sign of ill omen disappears permanently and the patient remains well indefinitely Although there are many exceptions, in general it is true that when nocturnal dyspnea Cheyne Stokes breathing pulsus alternans, gallop rhythm or block of one of the branches of the bundle of His occurs the prognosis must be guarded

Thyroid heart disease is unique in that the prognosis almost invariably is much better than it would be with the same apparent evidence of cardiac embarrassment if there were no hyperthyroidism When the diagnosis of hyperthyroidism and heart disease is properly made and effective treatment is instituted, patients who otherwise could rightly be expected to be incurable and even helpless invalids can generally be restored to comparative comfort or complete health There is no other condition in which such extraordinary improvement occurs and is so well maintained as in hyperthyroid heart disease For this reason the diagnosis is most important, although it is still too frequently overlooked It must be said, however, that the apparently sick patients are those who generally, but not *invariably*, *have an additional and independent form of heart disease* as well, such as mitral stenosis, hypertension or coronary artery disease and that even here the prognosis is still very good

In contrast to hyperthyroidism, the presence of chronic nephritis in a patient with heart disease makes the prognosis much worse Quite often when the term cardiorenal' is used in describing the clinical condition, there really is no significant nephritis The urinary findings are those of passive congestion and not of true renal insufficiency It is quite important to make this distinction from the point of view of prognosis and, ordinarily, simple methods are sufficient to indicate the proper differentiation Even when the urine contains albumin and casts, if the specific gravity is high the urine is of high color and there is no secondary anemia it is extremely unlikely that the kidneys are involved to any important degree Renal function studies may also be somewhat depressed as a result of passive congestion and return to normal when the cardiac condition improves It will be helpful, therefore, to appraise the renal factor intelligently in order to make a proper prognosis when there is congestive heart failure

There are other rarer forms of heart disease in which the prognosis may be excellent despite a considerable degree of heart failure, because treatment is so effective This applies to cases of beriberi heart failure from arteriovenous fistula, constrictive pericarditis, and heart failure accompanying acute nephritis or toxemia of pregnancy

Finally, in judging prognosis there are several types of complications that occur in heart disease which may be regarded as unexpected accidents which may suddenly change the outlook entirely It often is impossible to predict which patients will develop these complications or when they will occur There are certain general principles, however, that enable us to divide our patients into those who are more likely and those who are less likely to have these unexpected "accidents' I now refer to the development of subacute bacterial endocarditis, emboli (pulmonary or arterial), the different forms of paroxysmal rapid heart action, heart block and coronary thrombosis It is obvious that in a patient with heart disease who has been progressing most satisfactorily, the prognosis might suddenly become grave if one of these complications developed

As to subacute bacterial endocarditis, one may expect that patients with

well-marked mitral stenosis or hypertension as a group will only rarely develop it If there has been persistent auricular fibrillation or a past history of congestive heart failure, it is extremely unlikely that the patient will ever have subacute bacterial endocarditis, no matter how long he lives It also may be said that if there is no heart murmur, the patient not only has no subacute bacterial endocarditis but will not develop it in the immediate future On the other hand, those most susceptible to this disease are the patients who have been fairly strong comparatively free from dyspnea or recurrent attacks of rheumatism, able to work and who have shown an apical systolic murmur of mitral insufficiency or a basal diastolic murmur of aortic insufficiency In a word, it is the well-compensated patient with valvular disease, generally rheumatic having the above murmurs and a regular heart, without hypertension, who is most apt to develop subacute bacterial endocarditis

When it comes to predicting which patients will have emboli the problem is even more difficult In general it may be said that those with mitral stenosis are much more apt to have emboli either from the right auricle to the lungs or from the left auricle to the systemic circulation, than are those with aortic disease The presence of auricular fibrillation with or without mitral stenosis is also conducive to the production of emboli from either auricle Emboli from the ventricles (generally the left ventricle) occur almost exclusively following a coronary thrombosis and infarction of the ventricular musculature I know of no method however, of foretelling which ones of the above types of patients will actually have emboli When such an accident occurs the prognosis may need to be suddenly changed

The third type of accident in patients with heart disease is the sudden development of a new rhythm of the heart A patient with complete heart block and a slow rate of 30 to 35 may carry on in good health for a great many years Attacks of Adams-Stokes syncope however, do occur unexpectedly and any one of the attacks may be fatal It is impossible to predict when this may take place A much more common 'accident in which the rhythm of the heart changes is paroxysmal rapid heart action This may be due to auricular fibrillation, auricular flutter, auricular tachycardia or ventricular tachycardia This is not the place to discuss the differential diagnosis of these arrhythmias except to state that in most cases ordinary bedside methods suffice for their proper differentiation When the heart suddenly becomes rapid the clinical condition often quickly changes for the worse, but the patient generally does better than the physician anticipates because many of these disturbances are transient and self limited or respond most satisfactorily to treatment Furthermore there are many individuals who have no heart disease whatever who suffer from these paroxysms of rapid heart action and carry on indefinitely with practically no restriction in activities The important point with regard to these paroxysms is that the outlook is not so grave as it seems for most of them respond to proper treatment

Finally we must consider angina pectoris and the accident of coro-

nary thrombosis There is no other condition in the practice of medicine in which it is so difficult to prognosticate Patients who suffer from angina pectoris may die suddenly and unexpectedly shortly after they are first seen or may live for over twenty years Such sudden fatalities may but need not be the result of an acute coronary thrombosis In some the vessels, though showing atheromata, will not be thrombosed It is even believed that sudden death may occur in angina pectoris with a normal heart and normal coronary arteries If this does occur, it must be rare Patients with angina are also prone to attacks of coronary thrombosis that do not cause sudden death I know of no way of predicting when these accidents will occur Furthermore, when a patient has an attack of coronary thrombosis there is no satisfactory method of foretelling which individual patient will survive and which will succumb I have seen patients who were most seriously ill recover and others, who apparently were doing extremely well, suddenly die The age at which other members of the family died of coronary disease at times is helpful in prognosis Here the physician should remain hopeful under the darkest circumstances and yet give a guarded prognosis when the progress seems most favorable

Finally it is well to realize that as a result of improvement in the care of the cardiac patient, especially the proper use of mercurial diuretics, salt restriction and other procedures now employed, patients with congestive heart failure live a good deal longer than they did a generation ago We now see cases of congestive failure carry on for years who formerly died in months Some of the older statistics of prognosis, therefore, need revision

The foregoing are some of the general principles that I believe will prove valuable in formulating the prognosis in individual patients suffering from cardiac disorders It is clear that much remains to be known but I have found that these generalizations have been extremely helpful and that their use will at least diminish the number of errors in prognosis, which errors are constantly placing the medical profession in disrepute in the minds of the general public and encouraging the work of the irregular pseudomedical cults that pervade our country

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Nature and Treatment of Congestive Heart Failure

It is no simple matter to analyze in detail the nature and exact mechanism of congestive heart failure Despite exhaustive clinical, physiologic and pathologic studies there are many questions that remain unanswered It may be of some value however, to review some of the more generally accepted concepts that bear on this subject

Peripheral Circulatory Failure

At the outset the physician must clearly understand what conditions are not heart failure The fact that the heart becomes rapid and the blood pressure falls does not necessarily mean that the heart is failing or that there is any heart disease Patients who die of infections such as pneumonia, puerperal sepsis and typhoid fever die when the heart stops but death is not due to heart failure The enfeebled state of the circulation is due to peripheral circulatory failure Measures ordinarily employed to help the heart are generally useless and at times even harmful In this condition the circulating blood volume diminishes, there is an inadequate return of blood from the periphery due to stagnation or pooling of blood in the capillary bed and venules The heart is not apt to dilate and unless there are pulmonary complications the patient has no dyspnea, orthopnea or significant tissue edema

Peripheral circulatory failure or shock is common in infectious diseases and is often the presenting problem following hemorrhage, severe trauma burns, surgical operations and anesthesia and in stuporous states such as diabetic coma and cerebral hemorrhage Generally, patients with peripheral failure will show a cold, moist skin with blotchy cyanosis rapid pulse and an anxious facial expression The blood pressure often is low but the systolic level may at times be fairly well maintained while the diastolic level falls The cause of this state is not altogether clear, but that the primary difficulty in the circulation lies in the capillaries and the small peripheral vessels seems likely Studies have shown that the minute output of blood from the heart in this condition is apt to be lowered, and that the venous pressure is decreased rather than increased

As for treatment, digitalis is for the most part useless and probably harmful It will not slow the heart rate under these circumstances and

there is reason to believe that it will actually further diminish an already decreased cardiac output In fact, none of the drugs we have are of great value It would be desirable to increase the blood volume For this purpose injections of 1000 cc or more of 5 per cent glucose in normal salt solution may be given subcutaneously or intravenously Sometimes transfusions of blood are indicated The most effective method at present is the intravenous injection of plasma or human albumin For acute emergencies caffeine sodium benzoate (0 5 gm or 7½ grains) or coramine (1 to 5 cc), administered intramuscularly or intravenously, may be employed Inhalation of oxygen may also be helpful There may be rare occasions of acute circulatory collapse in which such heroic intravenous medication will be helpful and possibly life-saving There is hope that some of the newer preparations will be more effective in combatting the low blood pressure of circulatory collapse For no matter what the cause, a maintained low blood pressure is dangerous in that it sets up a vicious circle from which recovery is extremely difficult if at all possible Oliguria, nitrogenous retention and increasing stupor quickly result There is some evidence that preparations like paredrine (10 to 20 mg given subcutaneously) will favorably counteract this state and produce an elevation in blood pressure without an increase in the work of the heart

THE NATURE OF CONGESTIVE HEART FAILURE

Symptoms and Signs of Congestive Failure

It is only necessary to mention briefly the clinical evidence of congestive failure, as it is fairly well understood by most physicians One should distinguish failure of one ventricle from that of the other, although both are frequently involved Furthermore, it must be appreciated that subjective complaints generally precede physical signs as evidence of early heart failure Patients may show murmurs, arrhythmias and cardiac enlargement for many years and remain well compensated There are very few physical findings which of themselves would make one infer that heart failure is present or impending in the absence of symptoms Among these are chiefly Cheyne Stokes breathing, a diastolic gallop rhythm and possibly pulsus alternans, which may occasionally be elicited Most of the other physical findings follow subjective complaints

The earliest evidence of heart failure is abnormal breathlessness The patient notices shortness of breath on effort that formerly produced no distress This is due to failure of the left ventricle In some, dyspnea may come suddenly at rest, particularly at night The mechanism of these disturbances will be discussed With increasing dyspnea the amount of effort that can be tolerated comfortably decreases, finally reaching the point of orthopnea or difficulty in lying recumbent Cheyne-Stokes breathing is common In the earliest stages physical examination of the lungs may reveal no abnormality, although the vital capacity of the lungs will already be diminished and x ray may show some pulmonary congestion Later, increase in the pulmonary second sound, basal rales and finally hydro

thorax develop In some cases a slight unproductive cough coming at night or on effort occurs as a result of pulmonary congestion

The early evidence of right sided failure is increased venous distention or dependent edema The ankles begin to swell as the day progresses, the swelling disappearing overnight Later, edema of the legs persists and extends upward, involving the thighs, buttocks, abdomen and occasionally the upper extremities and face When there is considerable edema, it may involve the upper part of the body if the patient can lie flat, because of the effect of gravity As a part of the venous congestion due to right heart failure, the venous pressure increases, the jugular veins become distended and the liver enlarges Ascites may be present as a part of the general process of edema and partly as a result of portal obstruction Due to the congestion of the liver there is often epigastric pain and other abdominal symptoms In fact, occasionally pain in the right upper abdomen on walking may be an early or primary complaint, especially in patients with mitral stenosis, even in the absence of dyspnea or noticeable swelling of the legs In such cases the degree of activity is not great enough to elicit breathlessness, while the increase in right-sided failure is sufficient to cause pain in the liver

There are other general complaints that accompany heart failure but they are less distinctive and are met with too commonly in other conditions not associated with heart failure Weakness easy fatigability, insomnia irritability, indigestion palpitation and dull ache over the precordium have been emphasized by some authors as early evidence of weakening of the circulation Although these are complaints frequently found in cardiac patients, they rarely help in establishing the diagnosis of heart failure and in fact are often misinterpreted when due to other causes

The combination of both right-sided and left sided failure is seen more often than either one in its pure form Obviously all degrees of heart failure will be found In the advanced stage, cardiac patients become cachectic, showing marked loss of flesh and cyanosis of the skin, especially of the distal portions of the body such as the ears, nose cheeks hands and feet With this cyanosis there is often a slight icteric tint to the skin

Patients with marked congestive failure have impairment of sweating in hot humid weather In fact, they have been found to perspire only 25 to 50 per cent of normal and have much greater discomfort with such weather than other people However, in general, cardiac patients do better with moderately warm and dry weather than with cold

The Mechanism of Congestive Failure

For generations there have been two diametrically opposed schools of thought concerning the mechanism of heart failure According to one it is believed that heart failure is due to insufficient output of the heart to the tissues (forward failure) and to the other that it is due to congestion of parts of the body or back pressure (backward failure) The arguments pro and con will not be discussed here, but it may serve a useful purpose to mention some of the factors in circulatory dynamics that bear on this

question They will aid in understanding the events we witness in practice and in rationalizing the therapy we employ

One must distinguish some of the symptoms we often see in cardiac patients that may be looked upon as by products, complications or accidents of the condition and not as fundamentally related to the question of heart failure A patient may have attacks of Adams Stokes syncope and yet have no evidence whatever of congestive failure, not even experiencing anginal pain or dyspnea on the most strenuous effort Many cardiac patients suffer from weakness This may be due to many causes, among which a diminished cardiac output is important However, such weakness can occur without any congestive failure This is seen during prolonged paroxysmal tachycardia in which, because of an extremely rapid heart rate, the minute output of blood is markedly diminished The patient feels quite weak but he may have neither pulmonary nor peripheral congestion A similar situation in which there is marked weakness without congestive failure is seen in some cases of acute coronary thrombosis

It is of primary importance to look upon the left and right portions of the heart separately for, although we generally see the results of failure of both sides, it is often possible to distinguish the phenomena characterizing the failure of each side separately At times one will observe evidence of pure one sided failure The importance of a balance between the two ventricles becomes clear from the following analysis If, as a result of any cause, the left ventricle expelled one drop of blood less per beat than the right ventricle, within three to four hours about one liter of additional blood would accumulate in the lungs This would lead to acute pulmonary edema A condition similar to this occurs in acute left ventricular failure with paroxysmal cardiac dyspnea On the other hand, if the right ventricle expelled one drop less than the left an equal amount of blood would accumulate on the venous or right side of the circulation, resulting in venous distention, enlarged liver and, possibly, peripheral edema However, if both ventricles diminished their output equally, although the circulation would slow up there would be no accumulation on either side and neither pulmonary congestion nor peripheral edema would result In other words, although a slight imbalance of the two ventricles may exist for short periods of time, the two ventricles must eventually expel equal amounts of blood if disaster is to be avoided

The processes involved in the gradual development of congestive heart failure are both numerous and intricate Let us trace some of the events that occur during the progress of a case of mitral stenosis As the mitral valve becomes increasingly narrowed the pressure in the left auricle increases The wall of the left auricle becomes slightly dilated, which has two effects It tends to make the pressure within its cavity return to normal and, following Starling's "law of the heart," the slight dilatation improves the contraction and compensates for the obstruction of the valve The circulation is restored to normal but with the result that the left auricle is slightly larger, and eventually the pressure in the auricle

and the pulmonary circuit becomes increased As years go on and the condition progresses the right ventricle feels the burden of the increased pulmonary pressure The pulmonary second sound increases in intensity The right ventricle first dilates, thereby increasing its ability to expel blood, and later hypertrophies It is a matter of general experience that prolonged dilatation precedes and is the stimulus for hypertrophy In this way the bilateral balance of the two sides of the heart is maintained The equilibrium that now exists in this patient with mitral stenosis is such that, although there is no peripheral congestive failure, there has been a slight increase in the volume of blood in the lungs owing to the prolonged backward pressure behind the stenosed valve At this stage the patient may be short of breath on effort but shows no rales in the lungs The vital capacity of the lungs, however, may already have decreased This measurement often is the first evidence of a diminished cardiac reserve and is the result of a decrease in the total available air space in the lungs In the early stage, even if the vital capacity has not yet decreased when measured with the patient at rest, it will be found that effort will diminish the vital capacity whereas the same effort will produce no change in a normal individual

In following the developments of the above case the progression may take different courses If the right ventricle has become sufficiently hypertrophied and has not dilated too much, and the mitral obstruction is considerable, increasing breathlessness may result from further engorgement of the pulmonary circuit In fact such patients often have attacks of pulmonary edema At such times as a result of additional effort or of some nervous mechanism or for some other ill defined reason, the right ventricle pumps out more blood than the left and the difference is trapped in the lungs An adjustment of the circulation occurs as the right side decreases or the left side increases its output From this one can see how phlebotomy may occasionally be helpful in producing this adjustment This is the small group of cases with so called tight mitral stenosis with marked increase in pulmonary pressure, prone to attacks of pulmonary edema but without right sided failure who are now being subjected to shunting operations This will be further discussed later

The course of events, however, may be different The right ventricle may dilate beyond the point of mechanical efficiency With this there is an increase in the pressure in the right auricle, the venae cavae and the systemic veins This increase in the venous pressure (normal values are 40 to 80 mm of water) is the first evidence of right sided failure One of the earliest results of this is engorgement of the liver Another is the development of pitting edema of the recumbent portions of the body In some cases, despite the increase in venous pressure, peripheral pitting edema does not occur or is delayed because there are other factors concerned with the production of edema Among these one of the most important is the pumping action of muscular contraction of the legs on the venous return If there has not been enough dyspnea to prevent the patient from walking, the exercise of the legs tends to delay the develop

ment of edema Changes in the walls of the capillaries, the osmotic pressure of the blood, the development of acidosis, and to a lesser extent the question of tissue anoxemia, which is determined by the volume output of blood, all may have a bearing on the appearance of peripheral edema

The recent work of Stead and his colleagues has emphasized the importance of forward failure, mainly in the production of edema According to this concept, in the early stages of heart failure there is a decreased output from the left ventricle As a result there is a considerable decreased renal flow and retention of sodium This is interpreted as the primary cause of fluid retention and production of edema An important point in our new knowledge is that when there is a slight fall in left ventricular output (and even when there is no fall) there may yet be a considerable decrease in renal flow Different organs partake differently in the changes in blood flow, so that changes in total blood circulation cannot tell us what is happening in any particular organ

The above hypothetical case could therefore present itself at one stage as a patient with mitral stenosis complaining of dyspnea on exertion without physical signs of congestion but having a diminished vital capacity of the lungs At another time this patient might have a good deal of obvious pulmonary congestion with marked breathlessness or he might show considerable venous distention, enlarged liver, with or without pitting edema, and little dyspnea Finally, all the evidence of failure of both sides of the circulation may be present

If a hypothetical case had started with hypertension or aortic valvular disease the primary strain would have been on the left ventricle At first, because of the increased work, the left ventricle would dilate a bit, and thereby compensate to eject the normal amount of blood into the aorta If it did not do so for any significant length of time, acute pulmonary edema would result In the course of time the prolonged dilatation and subsequent hypertrophy of the heart progress beyond the point of improved efficiency and the left ventricle fails to propel as much blood as the right ventricle The result, as in the case of mitral stenosis, is increased pulmonary pressure, congestion of the lungs and dyspnea The right ventricle, laboring against a constant increase in pressure in the pulmonary artery, becomes hypertrophied This accounts for the frequent enlargement of the right ventricle that is found in cases of left ventricular strain Finally, when the right side is sufficiently embarrassed, peripheral edema, engorged liver and ascites may develop Patients who originally had primary left ventricular strain may be seen with various combinations of cardiac failure, some mainly with dyspnea, others with little or no dyspnea and showing for the most part peripheral congestion, and still others with different degrees of both

There are other effects on the dynamics of the circulation and physiologic functions of the various organs of the body that result from congestive heart failure The total volume of blood increases From a normal amount of about 5000 cc it may increase to 6 or 7 liters or more This

is one of the most constant changes that accompany heart failure This excess blood is stored throughout the venous side of the circulation, the lungs and to some extent in the dilated heart The fact is often overlooked that when a heart is considerably dilated there may be as much as 1 liter or more of blood in excess of normal within its chambers In such cases there must be a large amount of residual blood in the heart, for no increased amount is expelled with each systole The total blood volume will always decrease and approach normal with clinical improvement It has been difficult to explain what happens to the 1 or 2 liters of blood that disappear from the body as congestive failure improves Recently, however, it has been shown that during congestion there is an increase in red blood cell formation as evidenced by reticulocytosis, and with improvement there is a diminution in blood formation and an increase in blood destruction The latter is indicated by an increase in the icteric index of the blood and a marked increase in the urobilinogen in the stools The increase in blood volume in congestive failure is so constant that occasionally it may serve as a diagnostic point I recall an instance in which a man with definite organic valvular disease had a good deal of edema and pulmonary congestion There was ample evidence that he had nephritis as well At first the problem was regarded as one of heart failure It was found, however that the blood volume was perfectly normal This and other studies of the dynamics of the circulation all showed that the patient could not have heart failure and that the edema must be due to nephritis He died shortly thereafter of typical uremia

Disturbances in the velocity of blood flow are of considerable importance in cardiac patients In certain hyperdynamic states, such as obtain during fever, exercise, anemia and especially with hyperthyroidism, the rate of blood flow (not to be confused with the heart rate) is increased Blood moves more rapidly from place to place in the circulation With congestive failure the blood velocity is slowed up, mainly owing to the increase in the blood volume or circulatory bed When slowing is considerable there is likely to be pulmonary congestion, but slight slowing can take place in constrictive pericarditis, when the lungs are not congested One factor that has been neglected as causing a slowing in the velocity of blood flow as usually determined is the volume of the heart itself There are instances of marked dilatation of the heart especially of the auricles, when the cavities may contain 2 liters or more of blood The circulation time will necessarily be markedly prolonged because of the dilution in this enormous residual pool and the slow movement of blood, even if there is no pulmonary or venous stasis The methods in common use measure the time it takes for blood to flow from a vein at the elbow to the heart, through the lungs, out of the aorta to the tongue or respiratory center Normally this requires about fifteen seconds When delay occurs it takes place for the most part in the lungs or heart Patients with marked congestion may show a velocity of blood flow (arm to tongue) of twenty five to fifty seconds or more In fact if the rate of flow is normal it is unlikely that there is any chronic cardiac congestion of the lungs

There are various methods of measuring the velocity of blood flow that are in current use. Sodium dehydrocholate or decholin (3 to 5 cc. of a 20 per cent solution) may be injected into an antecubital vein. The time from the injection to the arrival in the tongue is signaled by the sensation of a bitter taste. Similarly, 2 to 3 gm. of saccharin in a few cc. of sterile water may be injected. The end point in this case is a sweet taste. Sodium cyanide (0.25 to 0.5 cc. of a 2 per cent aqueous solution) may also be used. The advantage of this method is that the end point is objective, for the patient will suddenly take deep gasping breaths the moment the drug reaches the carotid sinus. Alpha lobeline hydrochloride (5 mg.—0.5 cc. of 1 per cent solution) intravenously can be used. The end point is a sudden cough thought to come from carotid sinus stimulation. Calcium gluconate (2.5 cc. of a 20 per cent solution) may be used, in which case the end point is a sensation of heat in the tongue or mouth. Caution must be exercised in injecting calcium when the patient has been receiving full doses of digitalis because of a *synergistic action* of the two drugs. In all these procedures the patient should be recumbent and relaxed with the arm lying at about the level of the heart. The injection should be rapid and the end point accurately timed with a stop watch.

The preceding methods measure the time it takes blood to flow from a vein in the arm through the lungs, back to the heart and out through the aorta. If it is desired to measure the time consumed in the flow of blood through the lungs, the ether method is used. An injection of 0.3 to 0.5 cc. of ether with an equal amount of normal saline is made into the brachial vein. The moment the patient or the observer detects the ether in the breath is the end point. This time, which normally is about four to eight seconds, subtracted from the arm to tongue time is an indirect measurement of the pulmonary circulation time because most of that delay takes place in the pulmonary vessels.

Measuring the velocity of blood flow is a simple procedure for differentiating many conditions such as emphysema, bronchitis, bronchopneumonia, cancer of the lungs and aneurysms from heart failure. Not infrequently the features produced by these conditions, such as dullness, rales, dyspnea and chest pain, are mistaken for congestive failure. In all of these states the velocity of blood flow is normal or not much changed, whereas if heart failure is present it is slowed. It is evident, therefore, that a prolonged circulation time through the lungs is an indirect method *of detecting failure of the left ventricle. There is one important* exception to this general rule. When *acute* pulmonary edema develops in a patient who previously was in fairly normal health, as may happen with acute coronary thrombosis or in a well-compensated hypertensive individual, the velocity of blood flow may be essentially normal. It apparently requires a more chronic state of passive congestion in the lungs, so that pulmonary blood volume is markedly increased, before slowing of the circulation may take place.

An increase in the venous pressure likewise affords an early measure of failure of the right ventricle. This can often be estimated by observing

the degree of distention of the cervical veins Normally these veins are completely collapsed with the patient in the upright or semirecumbent position The height above the heart to which the distention rises serves as a rough measure of venous pressure The exact reading can be made directly from the antecubital veins (normally about 40 to 80 mm of water) In certain cases in which the question of congestive heart failure is in doubt, the determination of the venous pressure may be very helpful Obviously an enlarged liver or ascites may be due to a malignant growth alcoholic cirrhosis, inflammatory conditions or passive congestion Similarly, edema of the legs may be due to nephritis, varicose veins or cardiac failure In all such cases if the cardiac function is intact the venous pressure in the arms will be essentially normal

One of the simplest and most valuable of the measurements that aid in estimating cardiac efficiency is the vital capacity of the lungs Some of the factors that influence the vital capacity have already been discussed (See Chap 6) All that is needed to obtain this reading is a spirometer The apparatus is very simple inexpensive and requires no upkeep The test can be performed in one minute by any physician and will afford valuable information It has been very much neglected and should be used by all practitioners Once a diagnosis of heart disease has been made, the vital capacity determinations will be much more valuable in estimating prognosis and in following the progress of a cardiac patient than the more expensive electrocardiograms that are frequently taken Furthermore the finding of a normal or supernormal vital capacity practically rules out congestive heart failure This is particularly valuable in detecting cases of functional dyspnea when the readings are so often perfectly normal Diminution in the breathing space is a very early sign of left ventricular failure As has been stated this may precede the appearance of basal rales The decrease in the vital capacity results mainly from engorgement of the pulmonary vessels, which thereby diminishes the available alveolar spaces, and from a loss of elasticity of the bron chioles Other factors in some cases are the development of hydrothorax and even the huge size of the heart The volume of the heart occasionally may be actually 1 or 2 liters greater than normal It is obvious under such circumstances that, with the thoracic cage remaining unchanged in size, the available space for ventilation must diminish considerably Normally the average adult having a total breathing space of about 4000 cc inhales 400 to 500 cc with each breath and on effort can increase both the rate and the depth of respiration without distress When the vital capacity is diminished to 2000 cc he reaches his greatest possible depth of inspiration more readily and will resort to a greater increase in rate of respiration to attain adequate ventilation Furthermore, with these short and rapid breaths there is an increase in the proportion of the dead space (that upper part of the respiratory tract in which the air is not exposed to the blood for gaseous exchange) In this way a diminished vital capacity is conducive to dyspnea or uncomfortable breathing

It is also of some interest that pulmonary congestion may set up cer

tain vicious circles which tend to aggravate the very condition that produces them. Bouts of coughing, so common in cardiac failure, tend to increase still further the return flow of blood to the right side of the heart and thereby overburden the pulmonary circuit. The same is true of the act of overventilation or agitated breathing such as is seen during the period of hyperpnea of Cheyne-Stokes breathing. From this it is clear that whatever tends to quiet cough or vigorous breathing, such as a hypodermic injection of morphine, not only makes the patient more comfortable but actually improves the underlying congestion.

There is a form of breathlessness common in certain cardiac patients that is particularly prone to occur at night. It assumes forms of varying severity, from a slight increase in the degree of dyspnea to violent attacks of suffocation. It is often called "paroxysmal nocturnal dyspnea" or "cardiac asthma." It occurs most commonly in association with conditions in which there is a strain on the left ventricle, such as hypertension, aortic valvular disease and coronary artery disease. It is less frequent in cases of mitral stenosis. The factors that underlie this phenomenon are numerous. When the patient goes to bed there is a slight gradual shift of fluid from the periphery to the pulmonary circuit. Venous return from the legs becomes facilitated, blood volume increases through hemodilution, pressure in the peripheral veins and the right auricle rises and the right ventricle expels a little more blood than the weakened left ventricle can expel. There is also a slight increase in the metabolic rate of the body as the day progresses. This and the bodily activities during the day tend to produce greater pulmonary congestion. The vital capacity of the lungs which already had been diminished becomes further decreased. Furthermore, sensory perception is obviously diminished during sleep and the reflex stimuli from congested lungs, which are most important in the control of breathing, are less effective than during the waking hours. The result is that a greater degree of pulmonary stasis develops before the reflex stimuli from the lung are effective in producing exaggerated respirations. Finally the stimulus becomes sufficiently great to arouse the patient from sleep. The respiratory center now responds vigorously. The patient is then found to be breathing laboriously with evidence of slight or marked pulmonary edema. The cough that frequently accompanies the attack and the vigorous rapid breathing both tend to make the condition worse. The vicious circle may be broken by diminishing the return of venous blood to the right heart or by decreasing the sensitivity of the nervous system. The former is accomplished if the patient assumes the upright position or occasionally if phlebotomy is performed, and the latter by the use of morphine.

The foregoing discussion explains in general terms the mechanism of nocturnal dyspnea. There are, however, other less important influences involved which may serve as precipitating factors. The act of coughing, a full bladder, abdominal distention, unpleasant dreams, a change in environmental temperature, lowering the head or a large meal have all

been observed to precipitate attacks of paroxysmal dyspnea in cardiac patients. By some methods, probably through nervous reflexes, these factors serve as trigger mechanisms, but they all require a background of *pulmonary congestion to be effective in the production* of attacks of dyspnea.

Among patients who suffer from breathlessness, especially those with nocturnal dyspnea, and in some who are hardly aware that they have any dyspnea, periodic or Cheyne-Stokes breathing is quite common. Much attention has been given to its explanation but, although some light has been thrown on its mechanism, this peculiar phenomenon still remains obscure. It occurs mainly in patients with left ventricular failure, particularly in older people, and is most marked during, if not entirely confined to, the period of sleep. The breathing waxes and wanes with intervals of apnea varying in length from a few seconds to even a minute. The period of hyperpnea following the apnea may be very violent and awaken the patient in great agitation. In fact, this accounts for many attacks of nocturnal dyspnea.

Apart from the one predominant factor of pulmonary congestion there are other factors that have some bearing on the production of this type of periodic breathing. A diminution in the blood flow to the respiratory center, a decrease in the sensitivity of the respiratory center and of the entire nervous system during sleep, an increase in intracranial pressure and a diminution in the carbon dioxide content of the blood may all play their respective roles. Once a tendency to periodicity develops it is not difficult to understand why it may continue or become more marked. During the apneic intervals the carbon dioxide level of the blood (which is the main stimulus for respiration) increases, as carbon dioxide is not being expired from the lungs. The oxygen content of the arterial blood is decreasing, as very little oxygen is being absorbed from the lungs. After a sufficient interval has elapsed the stimulus becomes great enough even to arouse the insensitive respiratory center which responds in an exaggerated fashion. The result is violent hyperpnea which in time gets rid of the accumulated carbon dioxide from the blood through the lungs until the level is again too low to excite the respiratory center and the cycle begins again.

Morphine helps patients with Cheyne Stokes breathing not by doing away with the periodic breathing, for it may even lengthen the periods of apnea, but by diminishing nervous sensitivity and thereby preventing patients from being aroused by the uncomfortable hyperpnea. Caffeine in large doses, on the other hand, may temporarily eliminate this type of breathing by increasing the sensitivity of the respiratory center. More recently aminophylline given intravenously in doses of 0.24 to 0.48 gm (3¾ to 7 grains) has been found very useful. What is more important are the measures that improve the underlying pulmonary congestion. When cardiac therapy succeeds in producing a diuresis, sometimes even one of slight degree, the condition may be greatly improved. Furthermore, the

condition may be greatly helped by having the patient sit up in a chair with the legs hanging down rather than by keeping him in bed This also tends to diminish the venous return to the right heart

There are some other sequelae of congestive heart failure apart from those directly related to the dynamics of the circulation Fever and leukocytosis are commonly seen in conjunction with cardiac congestion In some instances an intercurrent infection may be the precipitating cause of the heart failure Frequently, however, congestion itself produces slight fever When the temperature is over 101° F it is more likely to be due to some infection like bronchopneumonia, rheumatic fever or sore throat, or to infarction in some organ, especially the lung The fact that slight fever (100 to 101° F) may result from uncomplicated heart failure, though commonly observed in ordinary cardiac patients, is best illustrated in rare cases of paroxysmal tachycardia Here one may see a patient without organic heart disease suddenly develop a rapid heart rate, and if the attack is prolonged he may gradually show evidence of pulmonary congestion and with it fever and leukocytosis With disappearance of the congestion the temperature and white blood count return to normal

Jaundice is another finding in some cases of heart failure There are three factors involved in the production of this type of jaundice In the first place passive congestion of the liver and anoxemia may impair liver function and disturb the excretion of bile through the bile ducts Secondly, pulmonary infarction is frequently associated with jaundice through the breakdown of red blood cells in the lung Finally, as congestion improves there is an increase in blood destruction The icteric index in some cases may reach levels as high as 20 to 30 or more As a result of prolonged hepatic congestion, cardiac cirrhosis may also develop This may produce the added picture of portal obstruction to the already existing cardiac edema In such cases repeated abdominal paracentesis may be required Ascites may be quite conspicuous and out of proportion to the other evidences of heart failure This occurs most frequently in cases of organic tricuspid stenosis and in constrictive pericarditis

In determining the presence or absence of congestive heart failure, it is helpful to distinguish the objective from the subjective manifestations Symptoms are more often misleading than signs Breathlessness may be functional or bronchial in origin Pain in the chest may be due to spondylitis and not to heart disease Likewise, the objective signs may also have other causes than the heart Peripheral edema may be due to varicose veins with its accompanying lymph stasis, rales to pneumonia, and enlarged liver to alcoholic cirrhosis or cancer It is often much easier to ascertain whether heart disease is present than whether there is heart failure

In observing and guiding the course of a patient with organic heart disease it is most important to bear in mind the factors that tend to precipitate heart failure Inasmuch as the underlying original causes such as rheumatic fever, arteriosclerosis and hypertension cannot as yet be prevented, care must be exercised in protecting cardiac patients against the

aggravating or precipitating causes The most important of these is infection The others are for the most part those situations that unduly increase the work of the heart Excessive physical effort, prolonged emotional strain obesity, pregnancy, anemia, hyperthyroidism or any condition that accelerates the heart may be contributing factors In so far as they can be prevented or remedied, just so far will ultimate heart failure be delayed

The distinction has already been drawn between congestive heart failure and peripheral circulatory failure It must also be made clear that both conditions may exist simultaneously This is particularly true in some cases of acute coronary thrombosis and when organic cardiac patients have intercurrent conditions such as pneumonia or cerebral hemorrhage When both types of failure coexist difficulties arise concerning some of the methods of therapy commonly employed A patient with severe acute coronary thrombosis may have pulmonary edema indicating left ventricular failure, and cold, gray, moist skin with a low blood pressure as a result of peripheral shock For the former condition phlebotomy might be useful and for the latter one might reasonably give a transfusion Likewise, digitalis is beneficial for the former and useless or harmful for the latter In such cases the physicians must weigh the advantages and disadvantages of the treatment in each individual instance

The Clinical Picture of Congestive Failure

A brief review of the clinical picture of congestive failure may now be summarized even at the risk of some repetition The type and severity of complaints and the variety of physical findings will depend on the particular case involved and the stage of the process that has been reached Symptoms as a rule precede signs Breathlessness is the most important and generally the earliest evidence of heart failure As has been discussed previously it is necessary to rule out other causes, such as those of a functional and pulmonary nature, before regarding breathlessness as due entirely to the heart In hypertension, aortic and coronary cases dyspnea may first appear at night, while in other cases it is first noted on effort Cheyne Stokes breathing especially during sleep almost always means heart failure Even before dyspnea occurs most cardiac patients complain of fatigue lack of pep, restlessness, insomnia and nervousness These general complaints are too common in many other conditions, particularly in neurasthenia, to be very helpful diagnostically

Edema of the ankles then develops At first it is transient, disappearing overnight Later it may persist When orthopnea does not prevent the patient from assuming the recumbent position at night, edema may spread upward earlier in the progress of the disease and involve the arms and face The liver becomes engorged and causes pain or discomfort in the upper abdomen, and in the course of time ascites may develop The amount of urine voided also decreases

Examination will show various findings in different cases Basal rales or diminution in respiratory excursion are among the earliest objective evidences of heart failure Diminution in respiratory excursion is reflected in

a diminution of the vital capacity of the lungs The x ray may show evidence of pulmonary congestion in the absence of rales Later free fluid accumulates in the pleural cavities, especially the right Auscultation of the heart may or may not reveal abnormalities depending on the nature of the lesion Murmurs, irregularities and even cardiac enlargement, if present, are not necessarily indicative of heart failure, though they may signify the existence of heart disease The presence of a diastolic gallop rhythm or pulsus alternans may generally be relied upon as signs of failure or impending failure

Cyanosis is apt to appear rather late In some cases of mitral stenosis and in chronic cor pulmonale, it is often very marked The cervical veins become distended and may remain so even when the patient is in the semirecumbent position Gradually, when severe heart failure continues for years, a high degree of cardiac cachexia with marked wasting of tissue takes place Such patients may show considerable swelling of the lower half of the body with very thin, emaciated arms and thorax The urine, which is small in amount, may show albumin and casts, but unlike that seen in chronic nephritis it will be highly colored and of a high specific gravity An unexpected gain of weight, while the food intake is actually small, often occurs and is due to the retention of fluid This may take place without obvious edema, for cardiac patients can have as much as 5 liters of excess fluid in the intercellular tissue spaces and yet show no pitting Slight jaundice, often due to a complicating pulmonary infarction, is not uncommon

Of greatest importance is the realization that most of the signs and symptoms of heart failure may be simulated in other noncardiac conditions An enlarged liver may be due to cirrhosis or cancer, pulmonary rales to pneumonia, bronchitis or tumor, edema of the legs to nephritis, pelvic tumor, varicose veins or hypoproteinemia, increased venous pressure to superior mediastinal obstruction, and so on One could further elaborate these similarities It obviously is necessary to appraise the entire picture most carefully in determining the presence or absence of congestive heart failure

The Action of Digitalis

There is hardly a drug in medical use that has been studied so extensively as digitalis Despite the most exhaustive pharmacologic investigations concerning its mode of action, many questions remain unanswered This discussion will be limited primarily to some of the known effects of digitalis and to the simpler aspects of its clinical indications

Digitalis in sufficient doses increases the irritability of the heart as indicated by the production of ectopic ventricular beats, first few in number, then in the form of ventricular tachycardia, and finally with the development of fatal ventricular fibrillation In the experimental animal the latter mechanism is generally the cause of death when lethal doses are given The therapeutic dose which averages about 35 to 40 per cent of the

lethal dose causes none of these irregularities except possibly a few extrasystoles

Digitalis also slows the conduction of impulses This is especially noticeable in a prolongation of the P-R interval The effect may be sufficient to result in partial heart block and rarely even in complete heart block Occasionally it disturbs the conduction of impulses through the main or finer branches of the bundle of His, altering the form of the QRS waves

Most of the slowing that results from digitalis and its allied drugs is produced by its stimulating effect on the vagus nerve Apparently some of the slowing is extravagal for it is only partly abolished by full doses of atropine In cases of auricular fibrillation, conspicuous slowing of ventricular rate results This is brought about mainly by the action of the drug on the junctional conductive tissue In hearts with a normal regular rhythm, only very slight slowing occurs This slowing is so slight that it cannot be the cause of such striking improvement as is witnessed in many cases of heart failure with a normal rhythm

There are other effects on the heart muscle produced by digitalis Possibly the most important pharmacologic effect of digitalis is that it increases the strength or force of contraction of cardiac muscle By direct action on heart muscle it lengthens the refractory period and slows the rate of propagation of impulses These effects are counteracted by the indirect action of the drug through the vagus which shortens the refractory period and accelerates the transmission interval The final effect is, therefore, variable, depending upon which influence predominates The drug has another direct effect on the ventricular musculature that is shown in a peculiar inversion of the T wave of the electrocardiogram (see Chap 21) It also causes a slight shortening of the Q-T interval Animal experimentation has shown that, while therapeutic doses of digitalis produce no pathologic changes in the heart muscle, toxic doses do cause definite necrosis of heart muscle fibers and inflammatory changes and also changes in brain cells Whether these findings are in any way related to the previously mentioned electrocardiographic evidence of myocardial effects is doubtful

It has long been known that digitalis diminishes the size of the heart This has been ascribed to an increase in tonus of the heart The possibility that this effect may be due to a peripheral mechanism was suggested by Dock and his coworkers They showed that digitalis produces constriction of the hepatic veins In this way it may be supposed that the drug pools blood in the liver and thereby diminishes the venous pressure and venous return to the right side of the heart, decreases the circulating blood volume and decreases heart size In any case, one of the most constant actions of digitalis from a practical point of view is that of decreasing the size of the heart

There has been much discussion concerning the action of digitalis on the coronary arteries and coronary blood flow Most of the evidence

points to a constricting action on these vessels due to a vagal effect It is unlikely, however, that this results in diminished blood flow through the coronary arteries because compensatory effects may result from the accompanying slowing and other actions of the drug

It is thought that digitalis improves the efficiency of the heart By this is meant that it enables the heart to do the same work with less expenditure of energy The evidence for this view is indirect and incomplete More knowledge concerning the intimate metabolism of heart muscle is needed It has been found that with heart failure the potassium and creatine content of heart muscle is diminished It is apparent that, when the heart is unduly dilated, a decrease in size brought about by digitalis might well increase the mechanical efficiency of contraction It can readily be seen that the effect of all these factors on the output of the heart will be variable The decrease in venous return would tend to diminish and the improvement in cardiac efficiency or capacity would tend to increase the cardiac output The former effect will predominate in cases in which the heart is normal or essentially normal and there is no abnormal residual blood in the heart, for no matter how efficient the heart may be it cannot pump out more blood than it receives When there is severe heart failure, however, the decrease in venous return is not a handicap and the improvement of the efficiency of contraction may be sufficient to produce an actual increase in the cardiac output Direct measurements, in fact, have shown that digitalis actually diminishes the output of the heart in normal individuals and in patients with conditions like pneumonia where the heart is regarded as normal In congestive failure, when the output may be either normal or somewhat diminished, digitalis has been found to produce a variable effect In many cases there is an increase but in some a decrease in the output depending upon which of the two opposite effects predominates

In the normal heart a small amount of blood still remains within the ventricular cavities after ventricular systole Emptying of the cavities becomes more complete with effort or when special demands are made In heart failure there remains a greater amount of residual blood after contraction, and when the need increases the ventricle involved (or both sides) is not able to keep pace and emptying of the chambers becomes even less well performed Digitalis aids the failing heart in performing this function more adequately

The main indication for the use of digitalis is congestive heart failure, whether it be left or right ventricular, or both It does not matter then whether the blood pressure is high or low, whether the heart rate is rapid or slow, or whether the aortic or mitral valve is involved When there is also a significant pulse deficit, as occurs in cases of auricular fibrillation or auricular flutter, apart from the previously mentioned effects, the drug improves the circulation by eliminating or diminishing the pulse deficit When there is considerable cardiac hypertrophy, slowing of the rate is more important because a thick musculature needs a longer diastolic rest period for oxygen diffusion It is open to question

whether or not it is desirable to give digitalis continuously to those patients who have no heart failure but who manifest conditions that might lead to failure, such as hypertension with cardiac hypertrophy If the circulatory dynamics are already normal and the patient has no symptoms it is likely that digitalis in therapeutic doses will upset the patient, and if there is no dilatation of the heart the cardiac output would probably be diminished by the drug rather than increased

Digitalis is also used for long periods of time in those patients who recover from heart failure, to maintain the improvement that has been obtained, whenever the underlying condition is chronic and it is feared that heart failure might recur There are disturbances in cardiac rhythm, not necessarily associated with congestive failure, for which digitalis may be used Paroxysmal or maintained auricular fibrillation or auricular flutter and instances of paroxysmal auricular tachycardia may often be helped considerably by the judicious use of the drug The rapid ventricular rate of auricular fibrillation or flutter may be slowed even when there is no heart failure and especially in the latter condition a normal rhythm is often resumed When attacks of paroxysmal auricular tachycardia recur frequently, constant administration of digitalis may prevent attacks entirely

It is important to emphasize that digitalis not only is not indicated but may be harmful in cases of peripheral circulatory failure In acute infections, surgical shock, heart tamponade from pericardial effusions or constrictive pericarditis, further diminution of cardiac output may result from the drug There are occasional instances in which the foregoing conditions are associated with true congestive failure and then digitalis may favorably affect the latter to a sufficient degree to be beneficial

THE TREATMENT OF CONGESTIVE HEART FAILURE

Unlike acute infectious diseases or many surgical conditions in which a complete cure is to be expected, the treatment of congestive heart failure is concerned with the amelioration of symptoms Its purpose is to diminish suffering, to prolong life and to increase the usefulness of the patient for as long as possible Although cures are not to be hoped for, except in a small number of cases, because the underlying structural changes in the heart are for the most part irremediable, proper treatment may render individuals more comfortable, may restore some to useful occupation and occasionally may secure even complete symptomatic recovery For the most part the best that is accomplished by intelligent care of a patient with chronic cardiac disease is a prolongation of life that compares very favorably with the advantages that are derived from the early diagnosis and treatment of cancer It is, therefore, with such restricted hopes that we must view this problem

At the outset, the physician must bear in mind that social and economic factors often enter into consideration in outlining a course of treatment This is as true of chronic heart disease as it is of many other chronic diseases A day laborer who suffers from recurrent asthma and hay f

cannot be sent on a sea voyage or to the mountains every so often to rid himself of the offending agents and to avoid exposure to them On the other hand, patients in more fortunate economic positions often derive considerable benefit from such expensive trips Similarly those patients with angina pectoris, who are in a position to afford it, can go south in the winter and avoid the burden of cold winters and inclement weather Likewise, the decision whether four or six weeks will be spent in the medical care of an individual case of cardiac failure may depend upon whether the man's job is jeopardized or whether he has sickness insurance, and other purely nonmedical considerations

Let us start with an average male of moderate means, 40 years of age, whose occupation is that of bank teller He has had mitral stenosis for many years and now presents himself in moderate congestive heart failure There has been increasing dyspnea, some cough and, finally, peripheral edema Physical examination shows moderate pitting of the ankles, a slightly engorged and tender liver, rales at the bases of the lungs and evidence of a slight right hydrothorax The heart is dilated, the action is absolutely irregular, typical of auricular fibrillation, with a rate of 130 and the murmurs indicate the presence of mitral stenosis The blood pressure is 150 mm systolic and 90 mm diastolic The Wassermann is negative and the other findings are not significant We may also assume that this patient previously had been receiving inadequate treatment and, in fact, had taken no digitalis whatever

Rest in Bed

The first principle in the treatment of such a patient is rest Some patients, especially men, will rebel at what appears to them as such an extreme measure They will plead to be permitted to cut down their work, to go into town for only one half a day or to remain at home resting or taking it easy' It is best at the outset to explain that more will be accomplished in a shorter time if a strict regimen is carried out An effective argument is to emphasize that they will start feeling better more quickly and will lose less time from work if they give themselves every advantage for recovery than if they go at it half heartedly I have found it helpful to explain that with *complete rest*, as compared with being merely ambulatory, the average heart saves about 25,000 beats each day and it is this enforced rest to the heart which is so beneficial The advice 'to go to bed' often carries with it a grave outlook in the mind of the patient However, the patient will have a better understanding of its significance and fear it less after a few days when he becomes aware of a distinct improvement It is desirable to obtain as much mental and physical rest as possible and for this at times it is wise to delay starting the entire course of treatment for a day or two so that the patient may take care of certain matters that would otherwise prey on his mind

When he takes to bed, simple devices should be used to make his bodily position comfortable The proper number of pillows or a back

rest, supports under the forearms and beneath the knees will aid considerably and often determine whether a patient will feel relaxed or restless. It may often be advisable to have him sit in a chair most of the day with the feet hanging down to prevent the shift of fluid from the periphery to the lungs. The use of a commode is often less of a burden than using a bed pan. During the early days visitors should be restricted, though diversions such as reading the newspapers and listening to the radio may be permitted.

I have become so convinced of the importance of the change in the dynamics of the circulation on assuming the recumbent position, which results in an increase in blood volume and an aggravation of the pulmonary congestion, that I now advise many patients to place wooden blocks under the head posts of their beds. These should be about 8 to 9 inches high and can be used indefinitely. This procedure is not particularly indicated when the main difficulty is hepatic congestion and edema of the legs or when the element of breathlessness is inconsequential. In some cases, moreover, it is better to have the patient stay in a chair all day. In fact, in recent years I have treated patients with congestive failure more and more by having them stay out of bed, in a chair, most of the day. The bed they use to sleep or rest in has the 9 inch blocks under the head posts so that at no time is the body really flat. They then do not need to use the bed pan at all and many are allowed to take some steps around their room. Patients are happier under this regimen than under the former method of strict bed rest, have fewer complications and, I believe, establish compensation more quickly.

Diet

There are some differences of opinion concerning the proper dietary management of patients with congestive heart failure. I have found beneficial the simple Karell diet consisting of 200 cc. of milk four times a day with no other food but allowing additional water up to 2 to 3 liters daily. This is particularly worthwhile if the patient is overweight and has hypertension. This diet is easily taken and is low in calories, salt and protein. It often produces a feeling of restfulness and relaxation which improves the general condition even without any other medication. An inanition diet like this may have beneficial effects because of the same factors that are at work in normal people living on a low caloric intake. It has been found that semistarvation produces a fall in blood pressure, in pulse rate and in the basal metabolic rate. This no doubt may diminish the work of the heart and may thereby improve the circulation when failure is present. Proger has even suggested keeping patients with severe congestive failure on diets of 500 to 800 calories for many months, allowing adequate protein and vitamins. The milk diet should be continued for two or three days or even longer. After this brief period a more general diet containing the proper amount of vitamins is gradually allowed, restricting the salt intake and limiting the fluids to about 2000 to 3000 cc.

effects It certainly is indicated in patients with dyspnea and with peripheral pitting edema of cardiac origin no matter what the circumstances may be There still is some dispute whether it acts beneficially in the presence of a regular heart beat Many of the English clinicians were of the opinion, and some still are, that its use is limited to auricular fibrillation In America it is generally accepted that, although the most dramatic responses are seen when auricular fibrillation is present, it has a decidedly beneficial effect even when the rhythm of the heart is regular A more detailed discussion of the action of digitalis was taken up at the beginning of this chapter

In using digitalis at the present time the physician is confronted with a great many different names and types of preparations Over thirty years ago pharmacologic studies showed that many of the preparations that were customarily used were below standard potency In fact, some were found to be almost inert The present situation is quite different Now they are practically all satisfactory and carefully standardized by the manufacturer The physician should choose a preparation that is not expensive for it may need to be used for a long time He should not be misled into believing a special pill or a tincture will not produce nausea They all can if they are active The matter of preference for pills or liquid preparations depends a good deal on local habits In England the tincture is still used a great deal whereas in America a pill or tablet of powdered digitalis leaves is more popular The latter in general is preferable because of the difficulty in controlling accurate dosage if the tincture is used It does not matter if the exact amount of sodium bicarbonate or many other medicines that are given is not known accurately, but it is imperative to know just how much digitalis is administered to our patients There still prevails a misconception that 1 drop of tincture of digitalis is equivalent to 1 minim I had the opportunity of looking into this matter during the First World War when it was my duty to standardize the digitalis preparations that were used in the American Expeditionary Force in France I found that the number of drops necessary to make 1 cc of tincture of digitalis varied from 30 to 60 These variations depended on the size of the dropper, the speed with which the drops fell and the angle at which the dropper was held It is apparent from this that when a patient receives 5 drops three times a day, as is still prescribed by some practitioners, he may be receiving very inadequate doses For these reasons and because of the convenience of administration, a pill containing 0 1 gm or 1½ grains of digitalis leaves (one cat unit) has proved to be a most suitable preparation Occasionally one may suspect that because the pill is very hard it might not be digested in the alimentary tract (intact pills have been found in the stools) Under such circumstances a tincture may be used At times one wonders whether the condition of the patient is not so low but that a pill would fail to be disintegrated in or absorbed by the alimentary tract Here also one may properly try a liquid preparation Finally individual patients occasionally find that they have less local irritation of the stomach taking one type than another type of digitalis

Opinions still prevail that certain digitalis preparations will be beneficial when others are not Careful studies, however, have shown that the margin of safety, i e , the difference between the therapeutic and toxic or lethal dose, is essentially the same for preparations like digitalis, digitoxin, lanatoside C, strophanthin or ouabain when given intravenously Confusion has resulted because absorption of these preparations varies considerably when given orally Ouabain, a pure crystalline glucoside obtained from *Strophanthus gratus* is absorbed very poorly from the gastrointestinal tract but is suitable for intravenous use (0 25 mg once or twice the first day and once daily thereafter) Strophanthin, a purified mixture of glycosides derived from *Strophanthus kombé* is similar in its action to ouabain although the dose is about twice as great Similarly, lanatoside-C (*Digitalis lanata*) is useful for parenteral use, the full digitalizing dose intravenously being 1 5 to 2 0 mg The oral dose, however, is much greater Six to 8 mg given in divided doses during two to three days will be adequate and the daily maintenance dose is about 1 0 mg (2 tablets of 0 5 mg) On the other hand, a preparation like digitoxin (a pure active principle of *Digitalis purpurea*) is completely and readily absorbed by the gastrointestinal tract so that the digitalizing oral and intravenous dose is practically the same, i e , 1 25 mg It is of some interest that this dose is only 3 cat units and is equal in potency to 15 cat units of the tincture or the whole leaf when given orally The reason for this discrepancy is that it is all absorbed whereas about four fifths of the ordinary digitalis given by mouth is nonabsorbable When digitoxin is used the full therapeutic dose of 1 25 mg may be given orally in one dose and then continued with a daily maintenance dose of 0 1 to 0 2 mg

During the past five years or so digitoxin has become increasingly popular in this country It has been used extensively in France for many years It has the advantage that it is completely and readily absorbed when given orally and that being a pure chemical it does not need to be standardized biologically Its potency is therefore constant It also produces much less nausea when therapeutic doses are administered, for the dosage by weight is about one thousandth of that required when digitalis leaves are employed There is one disadvantage, however Because early nausea is less likely to occur one may more readily exceed the therapeutic dose and inadvertently produce toxic results Another preparation that is being used is digoxin a pure crystalline substance derived from *Digitalis lanata* The methods of administration and the mode of action are somewhat like those of digitoxin The dose, however, is slightly greater, i e 1 to 2 mg for a single rapid oral effect and 0 25 to 0 5 mg as the daily maintenance dose It is rapidly absorbed and rapidly eliminated This drug can also be used intravenously in the above doses

Let us now return to the treatment of the patient mentioned above and let us assume that he had not received any digitalis before The average adult will require about 2 gm or 30 grains of digitalis or 20 cc of tincture of digitalis given over several days (The dose required in infants and children is apt to be one and one half to twice as great in proportion

to weight as in an adult) The exact amount will vary, some patients requiring less and some more This dose can be given quite rapidly, even within twenty four hours, but it is very rarely advisable to do so for it may turn out that only 1 5 gm or less were necessary and the remainder of the dose would have proved excessive and would produce toxic manifestations When the situation is very urgent and an effect is to be pro duced in hours rather than in days the intramuscular or intravenous route should be used instead of the oral There is no single method of dosage that needs to be followed Much will depend on the condition of the patient and the frequency with which he will be observed by the physician Generally it is satisfactory to give one fourth of the total dose or 0 5 gm the first day and a similar amount the second day One pill (0 1 gm each) five times a day will accomplish this On the third day the dose may need to be diminished If the apex rate, which was 130, has fallen to 100 it may be planned to give 0 1 gm three times on this day and after two or three days this may be cut down to one pill a day The hope is to lower the apex rate to 60 or 70 if possible, without producing any ill effects If this patient is to be seen only once a day this is best done in the morning, for then one can witness all the good or harm that the previous dosage has produced and one can outline the amount to be taken during the following twelve hours Finally the so-called "maintenance dose of 0 1 gm daily is kept up This is approximately the amount that is utilized or eliminated daily

The course outlined in the preceding paragraph needs to be modified under certain circumstances If the patient had received digitalis before it is better to start with smaller amounts One pill three times a day may then be sufficient On the other hand, he may be complaining of nausea and vomiting and doubt arises whether he has already received too much digitalis More often than not these symptoms are due to circulatory failure, generally accompanied by hepatic engorgement, and demand more rather than less digitalis If auricular fibrillation is present and the heart rate is still rapid one can feel quite certain that more digitalis is needed This same conclusion cannot be drawn if the rhythm is regular, for the rate then may remain rapid even after full doses of digitalis are administered Occasionally the electrocardiograms may be helpful in deciding the question although they are rarely necessary When nausea and vomiting are present and further digitalis is to be given the tincture can be used rectally Three or 4 cc (diluted in 50 to 100 cc of water) may then be given daily I have used this method with success and the vomiting has ceased even in patients who were convinced that it was produced by the previous digitalis that had been taken The dosage for rectal administration is the same as that for oral

When great speed of action is required some form of digitalis should be used intramuscularly or intravenously Such preparations are put up in ampules and generally contain 0 1 gm for each 1 or 2 cc Whereas most physicians are familiar with the proper oral dosage, the situation is

quite different with regard to the hypodermic use of the drug In fact the exact dose with the latter method is not so well known If 2 gm given orally is a proper digitalizing dose for a patient it certainly would be excessive if it were all given intramuscularly or intravenously in one injection On the other hand, 1 ampule containing 0 1 gm is often given hypodermically, especially by surgeons, hoping to give the patient the benefit of digitalis It is obvious that this dose is practically valueless The proper amount to obtain an appreciable therapeutic effect is between 0 5 and 1 gm given either intramuscularly or intravenously An effect may be expected with the former route in one to two hours and with the latter in fifteen to thirty minutes The same dangers exist, however, when digitalis is given intravenously as when strophanthin or ouabain is used, for the margin of safety between the minimal toxic and minimal lethal dose is the same for all these preparations The main danger is that if the patient has recently been taking any appreciable amounts of digitalis intravenous preparations should be given with great caution and in smaller doses

The following experience illustrates the occasional instance when rapid digitalization is imperative Some years ago I was called to see a woman about 40 years of age who was in a moribund state She had mitral stenosis with a regular rhythm and had been ambulatory and getting along very well She had not been taking digitalis That day at 9 p m she was suddenly stricken with palpitation and dyspnea and in a short time developed acute pulmonary edema When I saw her at 11 p m she was unconscious There was marked cyanosis, stertorous breathing and the lungs were full of moist rales The pulse was imperceptible but the heart rate as counted at the apex was about 190 and the rhythm was absolutely irregular She had already received strychnine camphor and caffeine hypodermically This was exactly the condition in which digitalis was indicated, but even minutes were precious I, therefore, gave her 8 cc (0 8 gm) of digitalis intravenously and in about twenty minutes the heart rate fell to 100 although the rhythm remained irregular The effect was most dramatic, the breathing quieted down and most of the rales disappeared A short time later the patient returned to her customary duties and presented the picture of a patient with well-compensated rheumatic mitral stenosis and auricular fibrillation

It is always important to watch for the indications to diminish or to omit digitalis The first obvious reason for cutting down the dose is if the desired therapeutic effect is produced In the case of the bank teller (cited above), if the heart rate slowed to 70 the dyspnea and edema disappeared and the patient was subjectively improved, the dose should be reduced to about 0 1 gm a day One, therefore does not need to push the dosage to obtain toxic effects if the therapeutic result is satisfactory The second reason for omitting the drug is if evidence of intoxication is detected This may be either subjective or objective It is curious how patients differ in their reactions to digitalis Some will quickly develop

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The course outlined in the preceding paragraph needs to be modified under certain circumstances If the patient had received digitalis before it is better to start with smaller amounts One pill three times a day may then be sufficient On the other hand, he may be complaining of nausea and vomiting and doubt arises whether he has already received too much digitalis More often than not these symptoms are due to circulatory failure, generally accompanied by hepatic engorgement, and demand more rather than less digitalis If auricular fibrillation is present and the heart rate is still rapid one can feel quite certain that more digitalis is needed This same conclusion cannot be drawn if the rhythm is regular, for the rate then may remain rapid even after full doses of digitalis are administered Occasionally the electrocardiograms may be helpful in deciding the question although they are rarely necessary When nausea and vomiting are present and further digitalis is to be given the tincture can be used rectally Three or 4 cc (diluted in 50 to 100 cc of water) may then be given daily I have used this method with success and the vomiting has ceased even in patients who were convinced that it was produced by the previous digitalis that had been taken The dosage for rectal administration is the same as that for oral

When great speed of action is required some form of digitalis should be used *intramuscularly or intravenously* Such preparations are put up in ampules and generally contain 0 1 gm for each 1 or 2 cc Whereas most physicians are familiar with the proper oral dosage, the situation is

quite different with regard to the hypodermic use of the drug In fact the exact dose with the latter method is not so well known If 2 gm given orally is a proper digitalizing dose for a patient it certainly would be excessive if it were all given intramuscularly or intravenously in one injection On the other hand, 1 ampule containing 0 1 gm is often given hypodermically, especially by surgeons, hoping to give the patient the benefit of digitalis It is obvious that this dose is practically valueless The proper amount to obtain an appreciable therapeutic effect is between 0 5 and 1 gm given either intramuscularly or intravenously An effect may be expected with the former route in one to two hours and with the latter in fifteen to thirty minutes The same dangers exist, however, when digitalis is given intravenously as when strophanthin or ouabain is used, for the margin of safety between the minimal toxic and minimal lethal dose is the same for all these preparations The main danger is that if the patient has recently been taking any appreciable amounts of digitalis, intravenous preparations should be given with great caution and in smaller doses

The following experience illustrates the occasional instance when rapid digitalization is imperative Some years ago I was called to see a woman about 40 years of age who was in a moribund state She had mitral stenosis with a regular rhythm and had been ambulatory and getting along very well She had not been taking digitalis That day at 9 p m she was suddenly stricken with palpitation and dyspnea and in a short time developed acute pulmonary edema When I saw her at 11 p m she was unconscious There was marked cyanosis, stertorous breathing and the lungs were full of moist rales The pulse was imperceptible but the heart rate as counted at the apex was about 190 and the rhythm was absolutely irregular She had already received strychnine, camphor and caffeine hypodermically This was exactly the condition in which digitalis was indicated, but even minutes were precious I, therefore, gave her 8 cc (0 8 gm) of digitalis intravenously and in about twenty minutes the heart rate fell to 100 although the rhythm remained irregular The effect was most dramatic, the breathing quieted down and most of the rales disappeared A short time later the patient returned to her customary duties and presented the picture of a patient with well compensated rheumatic mitral stenosis and auricular fibrillation

It is always important to watch for the indications to diminish or to omit digitalis The first obvious reason for cutting down the dose is if the desired therapeutic effect is produced In the case of the bank teller (cited above), if the heart rate slowed to 70, the dyspnea and edema disappeared and the patient was subjectively improved, the dose should be reduced to about 0 1 gm a day One, therefore, does not need to push the dosage to obtain toxic effects if the therapeutic result is satisfactory The second reason for omitting the drug is if evidence of intoxication is detected This may be either subjective or objective It is curious how patients differ in their reactions to digitalis Some will quickly develop

the subjective symptoms without the customary objective signs of intoxication and others do just the reverse There are still others who on very large doses develop neither

The most common early evidence of digitalis intoxication is loss of appetite, nausea and vomiting There is often a general mean and sickly feeling, accompanying the desire to vomit When this occurs there is no treatment except omission of the drug Other symptoms that are less common are diarrhea or yellow vision The objective evidence of intoxication concerns the findings in the heart Among these are undue slowing of the rate of the heart, i e , under 50, the development of extrasystoles, previously not present, especially in the form of coupled beats, and heart block The most common finding is digitalis coupling Here every second beat is a ventricular extrasystole This can easily be recognized if the rhythm of the heart was regular at the start but can also be detected at the bedside if auricular fibrillation is present Any of three forms of heart block may result, first degree (delayed conduction time), second degree (partial block) or complete block The first cannot readily be detected without an electrocardiogram, the second is easily recognized by noting an occasional dropped beat on auscultation, and complete heart block brought on by digitalis offers real diagnostic difficulties The ventricular rate of complete heart block produced by digitalis is not around 30, as it is in Adams-Stokes disease It is generally 60 or more and may be over 80 and 100 This mechanism is really an idioventricular rhythm in which the pacemaker for the ventricles is in the upper part of the auriculoventricular junctional tissue This regular rate will also continue to accelerate on increasing doses of digitalis In animal experiments this is one of the mechanisms by which death from digitalis intoxication occurs, although the more common mechanism is ventricular fibrillation As a practical matter, the first two types of heart block from digitalis do no harm and, in so far as slowing of the ventricle results, may be beneficial They serve merely as a signal that further dosage must be given cautiously Complete block, although rare, is an indication that the drug should be omitted or the dose should be diminished considerably Inasmuch as it may be difficult to detect this clinically it is well to omit digitalis if the heart, which was previously grossly irregular, becomes regular on digitalis, until the exact mechanism of the heart beat is known In such a case, regularization may be either due to a resumption of the normal rhythm of the heart, or the ventricles may be beating regularly while the auricles are still fibrillating In the first instance nothing would be lost by omitting digitalis for a few days and in the latter harm might result by continuing its use Electrocardiograms might be necessary in order that some of these unusual decisions may be made There are also electrocardiographic changes in the T wave of the electrocardiograms as a result of digitalis (Chap 21, Fig 168) which do not indicate intoxication, that occasionally may help in estimating the amount of the drug that a patient has received

Apart from digitalis or its allied drugs, like urginin, there is little else in the form of medication that acts directly on the heart, which one can

u e in the treatment of congestive failure Strychnine, camphor and caffeine had a considerable vogue in the past but there is very little satisfactory evidence to support their use except that caffeine may serve at times as a respiratory stimulus Adrenalin and ephedrine are useful in those cases in which the dyspnea or cough is in part due to an asthmatic or emphysematous state Potassium salts (such as potassium phosphate, 2 gm three times a day) have been recommended on the basis that there is a deficiency of potassium in the myocardium in cases of heart failure Much cannot be expected from their use Although many other drugs have supposed beneficial effects they prove to be hardly worth the trial

Diuretics

Next in importance to digitalis in the treatment of patients with congestive failure is the group of diuretic drugs Some of these are given by mouth and others are often administered hypodermically Among the former are theophylline (theocin) and theobromine sodium salicylate (diuretin) Diuretics are best given after complete digitalization has been accomplished, if there still remains evidence of peripheral edema It must be remembered that patients may have considerable latent edema in the body after all obvious pitting has disappeared Furthermore, when the subject has been in bed for some time, it is well to look for subcutaneous edema in the sacral region for fluid may accumulate there when the ankles are entirely free of edema Theophylline may be given in doses of 0 2 to 0 3 gm three times a day for one day and repeated every five to seven days if desirable effects result This may produce nausea and vomiting and when they are marked the drug should not be continued If an effect is produced with these doses it occurs within the first twenty four hours Sometimes several liters of urine will be voided in one day Following this there may be fatigue of the kidneys so that these large doses should not be repeated for about one week In ambulatory patients with a tendency to recurrent edema a single daily dose of 0 3 gm of theophylline is often well tolerated, together with the maintenance dose of digitalis, and produces just enough diuretic effect to prevent or postpone the necessity of abdominal paracentesis or intramuscular diuretics Theobromine sodium salicylate (diuretin) acts like theocin and also may produce distressing nausea The dose of this is 1 to 2 gm three times a day, once a week or 0 5 gm twice a day for a more moderate but continuous effect On rare occasions I have obtained a most satisfactory diuresis by giving 0 3 gm of theophylline intravenously well diluted in 10 to 20 cc of water This can produce nausea just as the drug does when given by mouth Similarly, aminophylline, either orally or hypodermically, has been used for diuretic purposes The oral dose is 0 1 to 0 2 gm three times a day, and the intramuscular or intravenous dose is 0 24 to 0 48 gm The difficulty is that in rare instances of coronary thrombosis fatalities apparently have been precipitated several minutes after intravenous injection of aminophylline However, aminophylline given in the form of rectal suppositories has considerable value, especially when paroxysmal

dyspnea is troublesome It then can be given each night or even twice daily When given intravenously its effect is quite prompt and beneficial in the acute stage of respiratory distress

During the past two decades mercurial diuretics have come into use and to a large extent are superseding those just mentioned Of these salyrgan, mercupurin, mercuxanthin and mercuhydrin are the most com monly employed They are given in doses of 0 5 to 2 0 cc intramuscularly, intravenously or by rectum in the form of suppositories If even a small amount of the drug leaks out of the vein or is given subcutaneously there is considerable pain and an ugly and stubborn ulcer may develop and last for many weeks Great care, therefore, must be exercised in adminis- tration There is less risk of any harmful effects if the diuretic is diluted with 10 cc of sterile salt solution when the intravenous route is used If the patient has veins that are difficult to find or if there is any doubt whether the needle is actually in the vein, it is better to withdraw the needle and give the solution intramuscularly I have had occasional ex- periences, however, in which it seemed that no diuresis resulted from intramuscular injections, possibly because the tissues were so edematous, when intravenous injections were very effective For the general prac- titioner the preferable method is to inject the drug deep into the gluteal region It should not be injected into the muscles of the arm This also is true for other intramuscular preparations, such as caffeine and digitalis, for soreness may develop in the arm and not in the buttocks The dose will vary and as often happens it may need to be repeated every few days to every few weeks for a long time Under these circumstances it may be necessary gradually to increase the amount I have seen a patient who had about 250 weekly injections over a course of five years always obtaining a most satisfactory diuresis of 5000 to 8000 cc in twenty four hours and during this time the individual dose was gradually increased from 1 cc to 4 cc At no time in this case was there any evidence of renal damage despite the enormous amounts of mercury that were used Very recently mercurial diuretics (thiomerin) have come into use that are safe and effective when given subcutaneously These may have the advantage that the patient or a member of the family may be taught to give the injection and thereby obviate the need of so many calls on the part of the physician The mercury suppositories that are available have been effective in some patients but in others they have caused distressing irritation of the rectum and had to be discontinued Within a few minutes after the intravenous injection of mercurial diuretics various types of cardiac arrhythmias may develop, particularly extrasystoles These are transient and harmless On very rare occasions sudden fatalities have occurred, mainly in cases of coronary artery disease

One would very much welcome a mercury preparation that would serve as an effective diuretic when given orally There are some available that are effective in occasional cases Such tablets contain 10 and 20 mg of mercury and about 40 mg of theophylline The dose is one to three tablets daily When three tablets daily are given for many days or weeks

renal impairment or other untoward effects may at times result It is safer to advise one or two tablets a day for a long period of time

In some cases the diuretic effect of salyrgan and mercupurin may be enhanced by the preliminary administration of ammonium salts Ammonium chloride in doses of 1 to 2 gm (15 to 30 grains) four times a day for about four days followed by the injection of 1 cc of the mercurial diuretic may be more effective than the mercurial diuretic without the preliminary ammonium salts In fact, occasionally a diuresis may be produced as a result of the ammonium salts alone The action is thought to result from the acidifying effect on the body These large doses of ammonium preparation are rather disagreeable but the annoying taste may be avoided if enteric coated tablets are given Another procedure is to give 2 0 gm of ammonium chloride two hours before each mercury injection and again two and four hours after the injection Ammonium chloride may be administered more or less indefinitely with the maintenance dose of digitalis in order to retard the tendency to recurrent edema

There are instances in which repeated doses of mercury have depleted the body of chlorides The chloride level of the blood will fall, the blood CO rise, and the urea nitrogen may increase At this point mercury injections may be ineffective and make the condition worse Mercury should then be discontinued and large doses of ammonium chloride orally, or even 10 gm in 500 cc of 5 percent glucose intravenously may again restore a more normal electrolyte balance and render the patient responsive to further mercury therapy

Two other methods of producing diuresis may be employed Two to 3 ounces (60 to 90 gm) of urea in a single daily dose over long periods of time can be used with beneficial effects Because of the nauseating effect of urea this will not be tolerated by most patients but for those who are not upset, it has proved of some value The disagreeable taste of urea may be somewhat disguised if it is dissolved in orange or grapefruit juice or in ginger ale It has also been recommended that all the sodium chloride be omitted from the diet and be replaced by 5 gm (75 grains) of potassium chloride daily I have occasionally found this to be quite useful though it may produce uncomfortable abdominal cramps

In general the various diuretics have added considerably to the treatment of patients with congestive heart failure When properly administered it may be expected that beneficial effects will result if the function of the kidneys is good At times it is no simple matter to anticipate without elaborate functional tests whether the kidney function is adequate or not Albumin and casts in the urine may result from passive congestion of otherwise fairly healthy kidneys as well as from true nephritis In general practice when it is not easy to perform chemical analysis of the blood the absence of any anemia and the presence of a high specific gravity of the urine points to a satisfactory functional state of the kidneys even when albumin and casts are present Only occasionally will it be necessary to determine the blood urea nitrogen or nonprotein nitrogen or to perform the phthalein test or a concentration test to ascertain more

accurately the state of the kidneys Even when it is found that the kidneys are slightly or moderately impaired, if there is congestive heart failure and edema persists despite digitalis and rest, diuretics may be used Although the results will not be so beneficial as when the kidneys are normal, such patients may yet do better following the use of mercurial diuretics than without their use

In the control of edema one of the most important factors is the retention or elimination of sodium For this reason a very low sodium intake is desirable, even as low as 200 to 500 mg daily It is doubtful whether it matters if this is obtained by an ordinary salt free diet or by the use of Kempner s rice diet Attempts are being made to eliminate sodium from the body, thereby obviating the need to institute such strict dietary regimens For this purpose cationic exchange resins, 100 gm daily by mouth, have been advised by Dock This abstracts sodium from the intestines but the medication is bulky and not very practical Another approach suggested by Schwartz is the use of sulfanilamide preparations which are carbonic anhydrase inhibitors These can produce an increase in the sodium output in the urine Unfortunately there are toxic side effects to this drug and therefore other nontoxic but similar preparations are needed

Cardiac cachexia and inanition are not infrequent results of long standing chronic heart failure and with this there may be an element of edema, purely as a result of the low protein content of the blood, just as occurs in nephrosis In fact, on occasions, because of the presence of considerable edema and certain other findings, a patient may be regarded as having heart failure when the edema is entirely due to nephrosis, nephritis, hypoproteinemia or some deficiency disease It is now well known that a fall in the total protein content of the blood serum may produce edema This is particularly true if the albumin content is markedly diminished, resulting in a reversal in the albumin-globulin ratio Such changes diminish the osmotic pressure in the blood and permit fluid to move from the blood into the tissue spaces Such hypoproteinemia may result from prolonged dietary deficiency, especially when the protein intake is restricted, from loss of albumin in urine and also from liver disease These factors not infrequently are involved in chronic cardiac patients The diets of these patients are often unwisely restricted by physicians or the patients do not eat because of persistent anorexia, and the liver is frequently in an unhealthy state from passive congestion The result is that some cardiac patients have edema not only because of decompensation but because of hypoproteinemia Examination of the blood must be made in all cases of stubborn edema to establish the correct diagnosis In some cases the edema will not be controlled until special efforts, such as the institution of a high protein diet or infusions of blood or, preferably, plasma, are made When intravenous injections are difficult or impossible, infusions of blood or albumin may be given intrasternally, an 18-gauge needle being used and the injection being given in the midline

Occasionally one observes patients in whom adequate response is ob

tained from the use of mercurial diuretics but who steadily lose ground As they become edema free their general condition grows worse Some seem to grow apathetic, drowsy and weak Although this complication has not been thoroughly studied it may be that when it occurs there is an increase in the renal insufficiency and dehydration of the tissues If this is so, some of the harmful effects might be obviated by increasing the amount of fluid in the diet When the kidneys are impaired in function and unable to concentrate well it requires a larger volume of fluid intake and of urine in order that the necessary waste products be excreted In fact, the prevailing custom of marked restriction in the intake of fluids for patients with congestive heart failure needs some revision because of this One other possible explanation of the ill effects that are occasionally seen following a brisk diuresis is that the fluids stored in the body may contain considerable amounts of digitalis and when this is absorbed into the blood stream for excretion through the kidneys the patient is really getting a new large dose of digitalis from within i e, he is being redigitalized Finally, disturbances in the sodium and potassium metabolism may prove to account partly for some of the ill effects following diuresis The administration of a pint of orange juice during the day the mercurial diuretic is given and again the following day has helped to decrease the ill feeling that some of these patients suffer This beneficial effect may be the result of the potassium contained in the orange juice, thereby making up for the loss of potassium during the diuresis

Fever is common with congestive failure and is an added burden on the heart Whenever this might be due to an infection responsive to specific therapy, such as penicillin or sulfonamides, appropriate measures must be carried out Occasionally it will be advisable to use a nonspecific antipyretic, such as amidopyrine (0 2 to 0 3 gm), for short periods of time to diminish the work of the heart

Since Dicumarol has come to be used so generally in thrombophlebitis and acute coronary thrombosis it also has been given to groups of cases of congestive heart failure Studies recently made, using the alternate case method as a control, have shown that those receiving Dicumarol during their hospital stay had a much lower mortality This was due to a marked decrease in thrombo embolic phenomena in those receiving anticoagulant therapy Therefore whenever feasible it would be advisable to give Dicumarol while treating patients for congestive heart failure

Mechanical Methods of Treatment

There are occasional instances of congestive heart failure in which it is observed that a patient, previously ambulatory and showing considerable edema of the legs but only slight breathlessness loses the peripheral edema after being put to bed but develops marked dyspnea As the right sided failure improves the left sided failure grows worse The recumbent position favors the return flow from the periphery, but improvement in the left ventricle is not sufficient to take care of the greater venous return from the right ventricle and so increased pulmonary congestion takes

place The fluid leaves the legs but appears in the pleural cavities and lungs The reason this does not happen more often as patients are put to bed is that the increased efficiency of the left ventricle keeps pace with the increased burden and a diuresis occurs The practical inference from these observations is that in many instances it is advisable not to keep the patient in bed but to allow him to sit in a chair with the legs hanging down It is much better to have fluid in the limbs than in the chest I have seen patients suffering from extreme breathlessness, despite oxygen therapy and all other customary methods of treatment, promptly improve and recover on getting out of bed into a chair In such a case one will observe dyspnea disappear while peripheral edema reappears The latter can be effectively treated later

In cases of acute pulmonary congestion in which circumstances might indicate the need of phlebotomy, tourniquets may be applied to the four extremities Pressure should be about 40 to 50 mm of mercury, or enough to prevent return flow of venous blood and yet to permit free forward arterial flow This traps the blood in the periphery and pressure may be maintained for a half hour to several hours using the following method Tourniquets are applied on three of the four extremities and every fifteen minutes one is released and applied on the limb that had been free In this way each extremity has a rest period every forty five minutes

Edematous fluid in the limbs can at times be removed by mechanical means Southey tubes may be inserted subcutaneously under aseptic precautions into the swollen feet or hands In this way several hundred cc of fluid or more may be lost daily and the edema thereby diminished I once saw a patient who lost over 15 liters of fluid in three days by this method

There remain several other procedures that are helpful in the treatment of patients with congestive heart failure It is not uncommon to forget that a diuresis may occur and the urine remain in the bladder This is particularly to be watched for in elderly males I recall the humiliation I experienced in finding 1500 cc of urine in the bladder when the postmortem examination was performed in a patient I had treated for heart failure The retention of urine in the bladder results primarily from prostatic obstruction Although patient and physician may have been aware of this prostatic difficulty before, at times the first significant evidence of it may occur during the bed treatment for cardiac failure It is obvious that the temporary remedy for this is catheterization

A more common complication is hydrothorax Most patients with advanced heart failure have some free fluid in the pleural cavities When the amount is small, i e, 100 to 200 cc, it is not detectable nor is it of any advantage to remove it mechanically However, when there is over 500 cc, or especially 1000 cc or more, considerable respiratory and general relief can be obtained by a thoracentesis The bases of the lungs, especially the right, should always be carefully examined in all cases Dullness or flatness on percussion, diminished breath sounds and decreased tactile fremitus with or without rales are the findings that help in detecting free

fluid at the bases of the lungs Tapping should be performed if it is expected to obtain more than 500 cc of fluid There is no advantage in withdrawing the very last amounts, and the needle should be removed when the fluid begins to come with difficulty or when an uncomfortable cough is produced It is always best to use a dull needle so that unnecessary scratching or bleeding of the pleurae will not take place

Abdominal paracentesis should also be employed when there is significant ascites Here also small amounts of fluid are better left undisturbed Tapping is indicated if the amount that might be obtained is more than 2000 cc The removal of smaller amounts does not seem to improve the condition sufficiently to make the procedure worthwhile Furthermore small amounts either in the abdominal or pleural cavities can readily disappear in the course of the routine treatment for heart failure, whereas the mechanical removal of larger quantities may expedite recovery When there is marked ascites, mercurial diuretics may be ineffective until the abdominal fluid is removed by tapping It would appear that the pressure of the fluid on the renal vessels prevented the mercury from producing its customary effect

Oxygen therapy may help patients with congestive failure This is of particular value for brief periods of time during cardiac emergencies when there is severe breathlessness and pulmonary congestion Positive pressure oxygen mask may be very beneficial in some cases of acute pulmonary edema Oxygen therapy is obviously not a procedure that is applicable in most cases of chronic failure which continues for months However, I observed a middle aged woman who had advanced irreversible congestive failure with striking cyanosis, dyspnea and a very large liver, carry on her work as a school teacher for a few years Apart from the customary cardiac medication she spent about two hours each day inhaling oxygen through a mask She was convinced that the oxygen enabled her to carry on

Finally a word must be said about phlebotomy As we all know, blood letting is an old method of treatment that was in vogue for centuries and used in all sorts of diseases Much of this practice has been discarded and I am of the opinion that at present its application in the treatment of congestive heart failure has not been sufficiently utilized In many of these patients if not in all, the total volume of blood is increased The various organs especially the lungs and the liver are markedly engorged The pressure in the venous side of the circulation is increased as is manifested by prominent distended veins in the neck and an increase in the venous pressure readings taken directly from peripheral veins There is both experimental and clinical evidence that after a phlebotomy the state of the circulation can be improved in some cases Venesection in severe hypertensive heart failure has been found to produce a fall in venous pressure and an increase in cardiac output, both favorable effects Some observations made years ago showed that the removal of 500 cc of blood from one arm improved the flow of blood in the other arm I have witnessed a decided decrease in the size of the liver and a prompt disappear

ance of pain and tenderness in that region directly after the removal of 700 cc of blood in a patient with mitral stenosis and auricular fibrillation What is much more striking is the effect of phlebotomy in some moribund cases The following experience illustrates very graphically some of the results that may occasionally be obtained by this neglected method of treatment I was once called to see a woman about 73 years of age who had asthmatic bronchitis, hypertension and myocardial failure All the customary treatment had been employed and when I arrived the patient was moribund, unconsciousness having gradually developed that day In fact, the breathing was such as is witnessed in patients only a few minutes before they die The chin dropped with each breath and there were tracheal rattles There was marked cyanosis and pulmonary edema The situation was so desperate that I did not even sterilize the needle that was used to puncture the vein About 600 cc of blood were removed in twelve minutes and just as the procedure was completed the patient became conscious In two weeks she was out of bed and the recovery enabled her to live in comparative comfort for another eighteen months In a second instance of this sort an elderly man who had been in a coma immediately regained consciousness after a phlebotomy of 550 cc of blood Here the children made the simple request that they wished to speak to their father once more He remained mentally clear that day, then relapsed into a coma and died the following day In these two and other similar cases the effect must have been a specific result of the bleeding because all other methods of treatment had failed and the improvement occurred literally minutes after the operation was started The amount of blood to be removed may be gauged by the level of venous pressure This should not be reduced below normal

The exact indications for phlebotomy are not clearly defined Beneficial results are not always obtainable even when the conditions appear to be similar The following conditions seem to be those in which bleeding may be helpful engorged or tender liver, distention of the veins of the neck, cyanosis and pulmonary edema When the blood pressure is still elevated under the above circumstances greater improvement may be expected from bleeding than if the blood pressure is low In fact, when a state of shock is present as in acute coronary thrombosis it may even be harmful If the onset of hepatic engorgement has been recent and acute as occurs shortly after the development of auricular fibrillation a definite diminution in the size of the liver may result from bleeding When the liver has remained congested and enlarged for months or years the secondary cirrhotic changes that take place prevent such a decrease from occurring after bleeding

A phlebotomy should be carried out very rapidly As large a needle as possible should be used and the entire amount of blood (400 to 700 cc of blood, depending on the size of the patient) ought to be withdrawn in about ten minutes The exact mechanism of the improvement that occurs is not clear Whether it takes place because of a diminution of the work of the heart by decreasing the volume of blood or by increasing the

'tonus' of the heart and thereby its contraction are matters that need not be taken up here In so far as it may be due to the latter it becomes desirable for the venous blood to be removed rapidly so that the dilated right side of the heart may decrease in size and regain a better tone before further blood returns from the periphery to redilate these chambers That this mechanism cannot be the sole one involved is illustrated by the beneficial results that occur from bleeding when acute pulmonary edema takes place in a patient with hypertension and sudden left ventricular failure Here there is no engorgement of the liver or appreciable venous engorgement This beneficial effect is probably the result of decreasing the return flow to the right ventricle and, therefore, the output to the lungs This enables a normal balance between the two ventricles to be established There obviously remain some important questions concerning this matter that need investigation in order that we may have more accurate indications and contraindications for the use of this valuable method of treatment

Recently instead of blood letting some have practiced the use of tourniquets on the four extremities These are applied so that the venous but not the arterial flow is impeded By this means blood is pooled temporarily in the periphery and prevented from returning to the heart

After several weeks of rest treatment, a patient such as we have described may be permitted to increase his activities At first he may be permitted to walk about in his room He then is gradually given greater liberties and finally starts going upstairs and out of doors When it is planned for him to return to his duties, it is well to have him begin by working only half a day Some restrictions in his activities must be enforced, as it is apparent that his circulation was not sufficient for the former expenditure of energy A conference between the physician, the patient and his family will help in ascertaining what part of his work should be curtailed, which entirely given up and which should be retained It is wiser to have him continue on only one half the previous amount of work with its decreased income, than to try to carry on full duties, knowing that in a short while he will again be bedridden Medical judgment tempered by good common sense is necessary in giving this advice

Quinidine

In the preceding discussion of the treatment of patients with congestive heart failure no mention was made of the use of quinidine This brings up a controversial subject and merits a separate analysis Quinidine was first proposed as a drug that would change auricular fibrillation to a normal rhythm It was accidentally discovered by Wenckebach who was told by one of his patients with auricular fibrillation that he could make his heart beat regularly by taking quinine It was later learned that quinidine was more effective in producing this change This was hailed as a great discovery because auricular fibrillation, once established or continuing for a week or more, was practically always expected to persist

indefinitely In fact it used to be called the perpetual arrhythmia Furthermore, it was also known that heart failure was often precipitated by and dated its onset from the change of the mechanism of the heart beat to auricular fibrillation It seemed reasonable, therefore, to hope that any drug which could keep the heart regular would be of great benefit The early reports concerning its use were very optimistic, but, as with many other therapeutic procedures, unfavorable aspects and limitations of its use gradually appeared and now it is known that the former enthusiasm was not altogether warranted However, there still remains a distinct field for its administration

In a sense, quinidine is a cardiac poison when given in sufficient doses It impairs the conduction of impulses and can actually inhibit contractions It lengthens the Q-T interval of the electrocardiogram, produces slight notching of the T wave and slows the auricular rate in auricular fibrillation The maximum effect in human beings occurs two to three hours after oral administration and parallels the degree of concentration of the drug in the blood stream There is only a slight amount of the drug in the blood at the end of twelve hours and only a trace after twenty-four hours The larger the oral dose, the higher the blood concentration There are some unexplained incompatibilities in the effects of quinidine I have seen cases of auricular fibrillation that have reverted to normal rhythm only four or six or more hours after the last dose Regularization must have taken place some time after the peak of blood concentration of the drug Furthermore, it is puzzling that quinidine can inhibit extrasystoles, tachycardia and fibrillation of the ventricle and yet at times produces these very same irregularities

The pharmacologic action upon which the action of quinidine depends in changing an irregular beat into a regular one, consists essentially of two factors It lengthens the refractory period of heart muscle and it also slows the speed of the cardiac impulse It will be recalled that auricular flutter and fibrillation are due to a circus movement of an impulse in the auricles This impulse keeps traveling around the vena cava at a very rapid rate (300 to 600), always finding cardiac tissue ahead of it that has already recovered from the refractoriness following the previous impulse and thereby permitting a continuous circus motion to persist indefinitely *If the refractory period of the muscle could be lengthened,* the impulse might find tissue still refractory and would stop The circus might be broken up in this way and this would permit the normal pacemaker with its slow inherent rate, which has been held in abeyance by the rapid rate of the circus, to start functioning On the other hand, if the rate at which the impulse travels around the circus is slowed or the path it takes is lengthened, it affords a longer time for the cardiac tissue to recover from its refractoriness and this would allow the circus wave to continue, thereby tending to perpetuate the circus One effect of quinidine (lengthening of the refractory period) would tend to break up auricular fibrillation or flutter and the other (slowing of the impulse) would tend to perpetuate it When the former predominates a regular rhythm is restored and when

the latter effect predominates the arrhythmia persists This explains the variable results obtained from the use of quinidine

Let us consider what might be expected from restoring a regular rhythm when auricular fibrillation is present In estimating the results of therapy both subjective sensations and objective changes must be carefully differentiated Many patients will say they feel much better after regularization, when on close analysis it will be found that they no longer are annoyed by palpitation The irregular heart beat is easily felt and causes not only discomfort but some apprehension They feel the heart less or not at all when it beats regularly, and believe that there must be great improvement because they know that a normal heart should beat regularly This does not necessarily mean that the heart is more efficient The best indication that the circulatory state is actually improved is the degree of dyspnea or the objective signs of congestive heart failure Patients with auricular fibrillation who have improved after quinidine may owe their recovery to the rest in bed and digitalis that was given during the period of observation To differentiate clearly the effects that are solely the result of quinidine, one should employ all the other therapeutic measures available for a few weeks and then, when the patient is in as good condition as possible, measure what further improvement may follow regularization When this is done the results will not always be favorable

Some years ago I undertook a study of this sort Inasmuch as dyspnea is the outstanding evidence of congestive failure and the vital capacity of the lungs is the best index of the degree of dyspnea, careful measurements of the vital capacity were made The vital capacity would increase as the condition improved following bed rest and digitalis therapy The heart rate would slow as was to be expected After the condition was stabilized quinidine was given When the rhythm became regular the vital capacity of the lungs showed no constant alteration—it was slightly less, the same or slightly greater This meant that breathlessness was not materially improved by regularization of the heart, if the rate previously could be adequately slowed by digitalis A heart that is beating irregularly at a rate of 60 to 70 can be about as efficient as if it were beating regularly at a rate of 70 to 80 In fact, a slow irregular rate may maintain a better circulation than a rapid regular one It was occasionally found that in the presence of stubborn congestive heart failure when the regular rate was 100 or more, improvement might first become manifest only after auricular fibrillation developed What would happen was that digitalis that was given failed to slow the regular rhythm and when auricular fibrillation began the drug slowed the ventricular rate to 60 or 70 and congestion began to clear In this sense the irregularity at times is an advantage

There are, however, some definite advantages of a normal rhythm over auricular fibrillation The formation of mural thrombi in the auricles with the possible development of emboli is much more likely when fibrillation is present Also, although the heart rate may be kept slow with digitalis when it is irregular, it accelerates less on effort when regular To these two factors may be added the subjective relief of palpitation

Taking everything into consideration, it is more desirable that the heart should beat regularly than irregularly Quinidine sulfate would, therefore, be a very useful drug if this change could be accomplished without risk However, there are dangers in its use Occasionally it causes sudden and unexpected death and it may result in the production of emboli The exact cause of death is not clearly understood It may result from an embolus but it would not be sudden under these circumstances I have had three unexpected fatalities in which postmortem examinations failed to show either emboli or mural thrombi A direct toxic effect on the heart may possibly account for some fatalities, as it is known that quinidine can inhibit the propagation of impulses Inhibition of auricular beats with temporary disappearance of the P waves has been observed following quinidine If the same type of effect is produced in the ventricles it would cause sudden arrest of the heart Another cause of death is respiratory failure In cats, this, rather than heart failure, is the primary cause of death from toxic doses of quinidine In fact, when respiration has ceased and the heart is still beating, recovery can result if artificial respiration is instituted and caffeine is administered intravenously even after a lethal dose of quinidine is given to the animal These experiments showed that the respiratory mechanism can fail while the heart is still viable and if respirations can be stimulated or maintained during the critical period for a long enough time, recovery may take place Such observations have a direct clinical application, for it follows that caffeine in large doses and artificial respiration may prove effective in tiding over some patients that manifest toxic action from quinidine

During the early years following the introduction of quinidine it was advised that in order to avoid unfavorable effects, patients with auricular fibrillation to be given this drug should be selected according to certain definite criteria It was thought that the most suitable were those in whom there was no great amount of enlargement of the heart, the irregularity was of short duration and there was no significant heart failure In other words, those with the least cardiac disability were regarded as the most favorable Unfortunately one of the most disastrous results in my own experience occurred when all these favorable factors were present This woman, about 35 years old, had a well compensated mitral stenosis and had been able to work steadily When the patient was first seen her heart was regular and three weeks later auricular fibrillation was found She came into the hospital for a tonsillectomy and it was decided that the heart should be regularized before the operation She was in very good condition and after a preliminary course of digitalis, quinidine was given The first day two doses of 0 2 gm (3 grains) were administered The second day the patient received 0 3 gm (5 grains) three times That evening the heart was regular, but there quickly developed marked breathlessness and the patient died in several hours I thought she had a pulmonary embolism from a thrombus in the right auricle Postmortem examination showed mitral stenosis and neither pulmonary infarct nor intra-

mural cardiac thrombosis It was after this experience that the animal experiments on the mechanism of death from quinidine were carried out Retrospectively it seems highly probable that this patient died of respiratory paralysis and that the same procedure that enabled the animals to survive (i e artificial respiration and caffeine) might have saved her life

Apart from the fatalities due to either cardiac or respiratory effects, emboli may become dislodged as the auricles stop fibrillating and start contracting regularly There is no known method of predicting in which case auricular thrombi are present and whether emboli will occur When they do take place embolic phenomena will develop within a few hours or days after the transition to a normal mechanism The emboli are more often arterial than pulmonary and may affect almost any part of the body Hemiplegia is the most common disabling complication Although such emboli occur in patients with auricular fibrillation who do not receive quinidine, they are much more common during this treatment and must then be regarded as the direct result of quinidine administration

Another limitation in the use of quinidine when organic heart disease (especially mitral stenosis) is present is that once a normal rhythm is established, in many cases after a short time there is reversion to the auricular fibrillation that existed before The regular rhythm is often maintained only for a few days or weeks and then the entire process of regularization needs to be repeated In many, it appears to be impossible or too difficult to keep the heart beating regularly Finally in only about 50 to 75 per cent of the cases will quinidine be effective in restoring the heart to a normal rhythm

Notwithstanding the hazards and limitations of quinidine it occupies an important place in therapy There are occasional instances in which it alone has been responsible for restoring compensation and one might even say in saving life The pros and cons must be weighed carefully and with increasing experience a proper selection of those cases in which its use is justifiable will result In the first place, from a review of my own experience and that of others it seems to be safe and effective when given to that group of fibrillators by no means small, who have no organic heart disease There is a considerable number who have this arrhythmia without other evidence of heart disease Here it may be expected that practically 100 per cent of the patients will promptly revert to a normal rhythm with only beneficial results As a corollary of this, it is very effective in the small number who continue to show auricular fibrillation two weeks or so after a subtotal thyroidectomy for hyperthyroidism when the basal metabolic rate has returned to normal It is useless to give quinidine before operation in such cases or even after operation if the basal metabolic rate has not been effectively lowered by the operation It is best to wait about two to three weeks after the operation, because in many cases spontaneous change to a regular beat will take place during this time In fact, if the change does not take place careful search for an undetected mitral stenosis should be made

The main problem concerns the use of quinidine when mitral stenosis, hypertensive or myocardial disease is present This comprises the group in which fatalities and emboli occur At the outset it must be appreciated that we have no certain method of avoiding these accidents The very early cases cannot be regarded as free from hazard, nor can the advanced cases be given up as hopeless There are some general principles that may guide us in these decisions All the disastrous results in my own experience have occurred in patients with mitral stenosis Although serious complications have been reported in nonvalvular cases, they are not so numerous I, therefore, have greater hesitancy in advising it in the former group When a patient can be restored to a favorable state of compensation on digitalis and the ordinary methods of treatment, it is doubtful whether quinidine should be used This is particularly true if mitral stenosis is present and the ventricular rate can be kept around 70 On the other hand, if the patient is doing poorly and it seems certain that he will not become ambulatory one would be justified in hazarding a course of quinidine Occasionally in cases in which there was apparently no hope, improvement has occurred Furthermore, when palpitation from the rapid irregular beat is a major and disturbing complaint the drug may be tried In some cases it is evident that as long as the heart was beating regularly, the patient was in comparatively good health and incapacitation dated from the onset of fibrillation When this disability has been present only a short while, one might risk quinidine therapy in the hope of preventing the slow downhill course that often follows the development of auricular fibrillation

Whenever it is decided to use quinidine for persistent auricular fibrillation, the patient should be treated in a hospital unless circumstances do not permit it The advantage of hospitalization is that changes in the mechanism of the beat may be more readily followed if electrocardiograms can be made whenever needed Ordinary methods of treatment including digitalis should be employed until as much improvement as possible is thereby obtained A maintenance dose of digitalis is continued during the period of quinidine treatment During the past several years, before quinidine is started and when time permits, Dicumarol therapy has been instituted The purpose of this is to diminish the likelihood of the development of peripheral emboli When the prothrombin time has reached the *desired level (35 to 40 seconds) quinidine is started The exact amounts* of quinidine that are employed and the speed with which the dose will be increased will vary with the urgency of the circumstances and somewhat with the custom of the physician The following is a routine course from which one can make individual variations as occasions arise The first day 0 2 gm is given at 10 a m , 0 3 gm at 2 p m and 0 4 gm at 6 p m The dose is increased by 0 1 gm each time, continuing the following days at the same hours of the day The patient is examined just before each dose to see if reversion has taken place If the auricular fibrillation persists the dose is increased If regularization occurs a maintenance dose

of 0 2 to 0 3 gm three times daily is continued for two to three weeks This dose is then decreased to 0 2 gm twice daily Whether quinidine therapy should eventually be discontinued or maintained and at what dosage can only be determined by trial and error and by experience in a given type of patient

The response of different cases varies considerably In some patients small doses suffice, in others single doses as large as 1 0 or 1 5 gm will be necessary for reversion Once the rhythm is regular, if doses larger then 0 3 gm three times daily are necessary to maintain a regular rhythm it generally will be better to discontinue quinidine therapy entirely and accept the fibrillation as permanent I recall an instance in which 0 8 gm three or four times a day was necessary to restore the heart to a normal rhythm and that same daily dose to prevent a return of the arrhythmia This was continued for months and enabled the patient to work, whereas otherwise he would have been bedridden with congestive failure

During the early days when increasing doses of quinidine are given the ventricular rate often rises while the rate of impulse formation in the auricles slows The acceleration of the ventricles is undesirable and palpitation becomes more uncomfortable This temporary aggravation of the condition is to be expected but must not be allowed to last too long In several days it must be determined whether the rhythm will revert to normal or not The drug should be omitted if any untoward toxic effects, especially syncope or marked acceleration in rate, develop When it is found that regularization does not occur, quinidine should be stopped entirely It should not be given in small doses like digitalis, for long periods of time because it will fail to restore a regular beat and only make it more difficult for the digitalis, which the patient is receiving, to keep the ventricular rate slow In other words quinidine is given to make the heart regular or to keep it so, but not for any length of time if auricular fibrillation persists

There is some reason to believe that quinidine might be useful in the prevention of auricular fibrillation in those prone to develop this irregularity One might expect that the hazards of its use would be avoided or at least diminished if it were given to some patients before fibrillation develops The difficulty is that there is no known method of predicting when this irregularity will occur and so one can rarely be convinced that the treatment is accomplishing its purpose Nevertheless, I occasionally advise patients with well marked mitral stenosis who are well compensated to take 0 2 gm of quinidine sulfate two or three times a day indefinitely in the hope that this distressing type of irregularity may be prevented

Quinidine has other uses than in the treatment of permanent auricular fibrillation It has been valuable in various forms of paroxysmal rapid heart action in preventing recurrences For this purpose it needs to be taken daily for long periods of time It is very effective in controlling ventricular tachycardia and often inhibits the occurrence of extrasystoles

of various forms These therapeutic problems have been taken up previously in other chapters and need not be considered here Suffice it to recall that it has an important place in the treatment of many arrhythmias

Surgical Procedures

The inadequacy of medical treatment for patients with chronic heart disease, just as in other fields of medicine, has impelled the profession to seek further aid from surgery The heart has been the last important organ to enter the scope of therapeutic surgery It is evident that operative work on the human heart will necessarily be difficult The circulation cannot be arrested for more than a brief time without sacrificing the life of the body even if the heart beat can be restored The brain in particular does not withstand anoxemia for more than several minutes Until a satisfactory artificial circulation is developed that will nourish the systemic organs and even the coronary arteries, any lengthy operative procedures on the inside of the heart will be difficult or impossible This is just as important as an apparatus for artificial respiration was for the development of pulmonary surgery Despite these handicaps, heroic attempts now and then are made to explore this new field Traumatic wounds of the heart are now successfully sutured Considerable progress in pericardial surgery has been made (Chap 5) Indirect methods of beneficially affecting the heart have been applied by altering the nervous system Cervical sympathectomy, dorsal ganglionectomy, and paravertebral alcohol injections of the dorsal rami and ganglia have been employed with some success in the treatment of angina pectoris (Chap 6) Daring attempts at incising and enlarging the orifice of a stenosed mitral valve have also been made When the first case of this sort was reported in 1923 by Dr E C Cutler and myself, we were hopeful that the operation of valvulotomy might prove useful because the patient survived and seemed somewhat better as a result of the operation The extremely high mortality in subsequent cases quickly placed this operation in disrepute

A simpler operation has been tried on rare occasions to give relief, when there is marked enlargement of the heart, i e, decompression of the chest Occasionally this has been done believing that pericardial adhesions are present only to find subsequently that the pericardium was normal Despite this mistaken diagnosis, clinical improvement has been noted in some such cases I had one experience in which the removal of a generous portion of several ribs overlying the precordium was of considerable benefit This patient was about 25 years old and had mitral stenosis, auricular fibrillation and marked cardiac enlargement The circulation was maintained in a fair state of compensation by the constant use of digitalis I had followed this case for many years The patient finally developed dysphagia which resulted from pressure of the enlarged left auricle on the esophagus This was purely a mechanical difficulty and did not respond to ordinary methods of treatment The dysphagia was promptly cured by removing under local anesthesia portions of ribs overlying the precordium Not only was the patient able to swallow nor

mally after this operation, but palpitation, which had been very annoying before, was much improved. She then did not feel the rapid irregular heart beat because it was no longer pounding against bony ribs but rather on a soft cushion of muscles and subcutaneous tissue. Even the ventricular rapidity seemed to be better controlled by digitalis than formerly. The patient lived for about nine years after the operation. Such decompression of the chest may be indicated whenever an enlarged heart is producing distressing symptoms as a result of pressure. This may not only involve the esophagus but also the left bronchus, causing harassing cough. Enlargement of the left auricle and of the pulmonary artery may also produce hoarseness or aphonia as a result of paralysis of the left vocal cord caused by pressure on the left recurrent laryngeal nerve. Similar operative procedures that decompress the chest may possibly prove useful in such conditions.

The hopelessness of many cases of advanced cardiac disease has been responsible for the development of another surgical procedure, complete thyroidectomy. Its application in the treatment of patients with angina pectoris has already been discussed (Chap. 6). It also has been performed for intractable congestive heart failure, due either to valvular or myocardial disease. It was known that when the basal metabolic rate is elevated as in hyperthyroidism, and there is congestive heart failure, a subtotal thyroidectomy with a subsequent fall in the metabolic rate caused the heart failure to disappear. It was also known that in myxedema associated with a sluggish circulation, congestive heart failure was very rare. It was, therefore, theoretically assumed that by producing myxedema in a patient without hyperthyroidism who has heart failure the demand on the heart might thereby be lessened to meet the supply. However, it was overlooked that in the production of myxedema, not only is the demand diminished by lowering the metabolic rate, but the supply is also depressed, for the volume output of the heart is diminished and the velocity of blood flow is slowed in spontaneous and artificial myxedema. If the circulation is improved by this procedure, therefore there must be other factors at work. The inherent metabolism of the heart may be altered so that it does its necessary work more efficiently and with less proportionate expenditure of energy. Another possibility is that with a partial myxedema, the heart is less sensitive to certain reflexes or internal hormones such as epinephrine. Finally a most important factor is the size of the heart. A certain amount of dilation of the heart may be beneficial but excessive dilation impairs the efficiency of the circulation. In congestive heart failure the heart is already dilated to a greater or lesser extent. When myxedema is produced, there is a tendency for further dilation to occur. May not improvement depend on this unpredictable factor, i.e., whether the further dilation of the heart is excessive or not? At any rate the pathologic physiology of this problem is still unsettled.

The clinical results of complete thyroidectomy have been variable. In those suffering from extremely advanced lesions, if improvement occurs it may not last long enough to warrant the operation. On the other hand,

the operation does not seem justified if the disease is only slight or moderate or if the disability is not great, because of the handicaps attending partial myxedema There remains a small group of cardiac patients, neither too sick nor too well, who may be suitable for this operation There is no doubt that some of the patients who had complete thyroidectomy have been improved as far as their symptoms of heart failure are concerned, but it is hoped that a simpler method might be devised to obtain similar results without producing the ill effects that follow the removal of an important vital organ This work must still be regarded as in the experimental stage, but has already served as a forceful stimulus to the exploration of new fields that might bring relief to those suffering from intractable heart disease At present I do not advise total thyroidectomy in the treatment of persons with chronic cardiac disease

Nonsurgical methods of decreasing thyroid function and producing a partial myxedema are now being employed The first consists of administering large doses of propylthiouracil to patients with intractable congestive failure or angina who have a normal thyroid gland This may need to be continued for several months or more before a lowering of the metabolism occurs The doses required are likely to be large—100 to 200 mg three times daily The second method is the use of radioactive iodine This is much simpler but at present can be given by only a few physicians under very careful supervision The early reports of Blumgart indicate that the results are fairly satisfactory in the majority of cases of angina but less so in those suffering from congestive failure The doses of radioactive iodine employed have varied from a total of 25 to 120 millicuries given in interrupted amounts at varying intervals No injury to other organs has been observed although in about one half of the cases a mild sore throat and cough has occurred which disappeared in a week or so The cholesterol was found to rise before the basal metabolism fell The results of these two types of treatment need to be observed more extensively and for a longer time before final judgment as to their value can be determined

Another surgical procedure that is being employed for hypertension and to some extent for hypertensive heart failure is sympathectomy Various types of operations are being tried, but it appears that the dorso lumbar sympathectomy of Smithwick is at present most promising More extensive division of the sympathetic nervous system, including all the dorsal and lumbar branches, may prove to be more effective Already many successful results have been obtained, even when myocardial involvement was present as shown by the presence of gallop rhythm and markedly abnormal electrocardiograms This approach to the problem of hypertension and its complications deserves our most careful interest and attention

There is one peculiar though rare form of congestive heart failure that responds most dramatically to surgical treatment, i e , the type following *arteriovenous aneurysm or fistula* As a result of a traumatic wound or less frequently following an infection, a communication may become estab

lished between an artery and an adjacent vein Blood is then shunted through the circulation and the work of the heart becomes definitely increased In the course of time dilatation of the heart, murmurs and congestive failure are apt to develop if the blood vessels involved are of significant size The diagnosis is easily made by the history and the findings of a continuous murmur and thrill at the site of the aneurysm with accentuation during systole Furthermore, compressing the fistula produces an immediate and characteristic slowing of the heart rate The surgical obliteration of the communication between the artery and vein can result in a complete disappearance of all subjective and objective evidence of cardiac failure

An arteriovenous fistula occasionally is discovered in the lungs The expected continuous murmur will be audible which resembles the murmur of patent ductus arteriosus At times the two conditions may be confused although an x ray finding of a shadow in the lung ought to be diagnostic Apart from the auscultatory findings this condition may be associated with cyanosis, clubbing and polycythemia It is curable by surgical operation

With the rapid advances that have taken place in surgery of the heart during the past decade or two it is not surprising that attempts at operations for mitral stenosis have been revived The early work by Cutler and myself some twenty-five years ago in which the valve was approached through the ventricle did not prove successful, although it opened the field of direct surgical approach to chronic valvular disease of the heart More recently, the mitral valve has been incised through the left auricle and the results are more promising Bailey performs a commissurotomy and believes that stenosis is at least partly relieved thereby without adding any significant degree of mitral insufficiency He has already obtained great improvement in some of his cases with a fairly low operative mortality Harken performs a finger fracture of the stenosed mitral valve, also approaching the valve through the left auricle From an experimental point of view, artificial valves have been devised to by-pass valvular obstructions in animals and have been found to function We all look hopefully for further perfection of surgical technic so that some of the hitherto incurable conditions may become amenable to treatment

Indirect surgical methods of relieving pulmonary congestion have also been tried In cases of tight mitral stenosis with a high degree of pulmonary back pressure and congestion but without right sided failure there are various means by which the congestion might be relieved One would be by making a window in the interauricular septum, thus imitating a Lutembacher syndrome This would relieve the pressure in the left auricle and thereby in the lungs, and would shunt the blood to the right side of the circulation This operation has already been performed by Harken Another procedure is to tear the tricuspid valves and produce tricuspid insufficiency In this way less blood would be expelled into the lungs The situation would somewhat resemble what obtains in cases of mitral stenosis that also have tricuspid stenosis Here there is much less

more slowly, about 200 mm per second It picks up speed as it continues into the auriculoventricular bundle but it is still conducted at a relatively slow rate The bundle runs forward for a short distance on the crest of

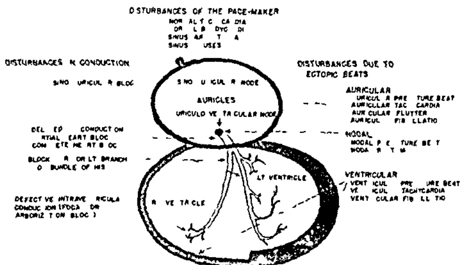


Fig 1 A schematic diagram illustrating the common types of disturbances in the mechanism of the heart beat

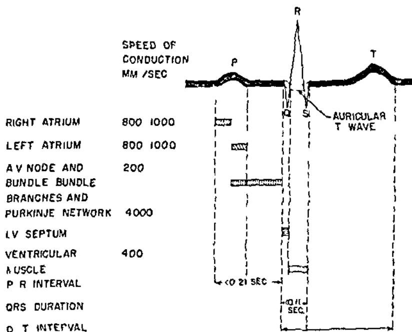


Fig 2 Speed of conduction in various parts of the heart and its representation in the normal electrocardiogram

the muscular portion of the interventricular septum at its junction with the membranous septum (undefended space), then divides into a right and left branch The impulse travels even faster in the bundle branches which course down on each side of the interventricular septum The

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dyspnea and pulmonary congestion (see Chap 4) This operation has been performed by Cossio of Argentina Another method also reported by the same observer consists of ligating the inferior vena cava below the renal veins, just as has been done in some cases of thrombophlebitis of the legs This would also decrease the return flow to the right heart and alleviate pulmonary congestion In the reported cases it has stopped the recurrences of acute pulmonary edema

A final technic reported by Bland and Sweet consists of suturing the azygos vein to one of the pulmonary veins, gapping the bridge with a vitallium tube I have seen two of their cases in which the results appear to be dramatic These shunting operations, particularly this last one, are primarily applicable to a selected group of cases The ideal situation is one in which the heart is not much if any enlarged, although the left auricle is dilated and there is a high degree of pulmonary hypertension The latter is reflected in the tendency to repeated hemoptysis or attacks of acute pulmonary edema In these cases catheterization studies show a marked increase of pressure in the right ventricle, pulmonary artery and pulmonary capillaries There should be very little if any evidence of right sided heart failure Although none of these operations attacks the underlying lesion of the mitral valve, and are therefore only palliative, it seems quite certain that they can afford relief of distressing symptoms Until more effective methods have been devised, there can be no doubt that they will have a useful place in properly selected individuals

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Clinical Electrocardiography

THE NORMAL ELECTROCARDIOGRAM

Normally the impulse that initiates the heart beat arises at the sino-auricular node of Keith-Flack a club shaped structure consisting of specialized tissue measuring about 1 5 cm in its greatest length and located high in the right atrium near the orifice of the superior vena cava This tissue is different in structure from the rest of the cardiac musculature and is rich in nerve fibers and ganglion cells Although there is reason to believe that rhythmic electrical activity must exist within this structure, thus far conclusive evidence for electrical activity within the node has not been demonstrated So far as we know there is no representation of sinus activity in the normal electrocardiogram The wave of excitation emerges from the sino auricular node into the auricular muscle proper Hence the node is called the pacemaker of the heart Since there is no specialized conduction path in the auricles, the impulse is then conducted radially over the auricles in much the same manner as a ripple spreads over the surface of a pond when a pebble is dropped into it Because of the thinness of the auricular walls the epicardial surface is activated as soon as the underlying endocardial surface This wave spreads over the auricles, depolarizing first the greater part of the right atrium then the greater part of the left atrium At or shortly before the height of the P wave the right atrium has been activated the left atrium is activated during the second half of the P wave The impulse is conducted over the auricles at a fairly rapid rate, in the neighborhood of 1000 mm per second The wave of depolarization is followed by a wave of repolarization (the so called auricular T wave) but this is ordinarily swallowed up in the succeeding ventricular complex of the normal electrocardiogram

When the first half or so of the P wave has been written and the right auricle activated the wave of excitation is picked up by the junctional tissue between the auricles and ventricles This structure is called the auriculoventricular node of Tawara which continues as the auriculo ventricular bundle of His This junctional tissue also has a specialized structure like the sino auricular node The auriculoventricular node is an ovoid structure located beneath the right atrial endocardium just below the orifice of the coronary sinus Here the impulse is conducted much

more slowly, about 200 mm per second It picks up speed as it continues into the auriculoventricular bundle but it is still conducted at a relatively slow rate The bundle runs forward for a short distance on the crest of

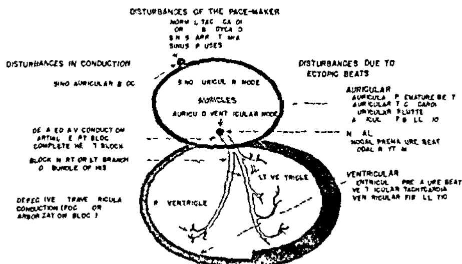


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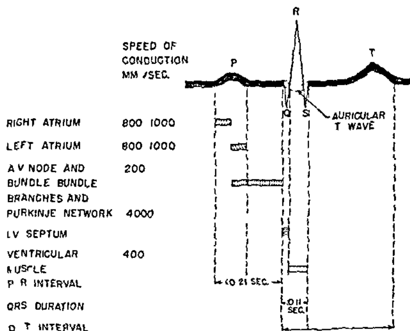


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the muscular portion of the interventricular septum at its junction with the membranous septum (undefended space), then divides into a right and left branch The impulse travels even faster in the bundle branches which course down on each side of the interventricular septum The

right branch becomes a part of the anterior papillary muscle of the right ventricle and does not branch again until the apical extremity of that muscle is reached (Fig 1) The left branch, on the contrary, in most if not all individuals, begins to branch shortly after it has become the left main stem These specialized conduction paths continue with finer and finer arborizations (Purkinje fibers) and spread throughout the two ventricles, being particularly abundant on the endocardial surface of the ventricles All of the electrical events described so far, activation of the auricular musculature, the auriculoventricular node, the auriculoventricular bundle, the bundle branches and the Purkinje network, are included in that part of the normal electrocardiogram from the beginning of the P wave to the beginning of the ventricular complex (Fig 2) However, the spread of the impulse through the main bundle branches and the sub endocardial Purkinje network is so rapid that these events have virtually no duration and are not represented in the normal electrocardiogram

Because of the earlier arborization of the left bundle branch, the left side of the septum for a brief instant, is activated earlier than the right Spread of the impulse in the muscular septum and the free walls of the ventricles is much slower, 400 mm per second, than spread in the Purkinje tissues which has been measured at 4000 mm per second The spread of the impulse is in the direction from the apex toward the base of the ventricles on the endocardial aspects of their free walls and approximately at right angles to the endocardium from the subendocardial network to the epicardium Because of the rapidity of subendocardial activation this process is almost synchronous throughout the ventricles As a result both ventricles are stimulated to contract almost simultaneously Actually, since the right ventricle is thinner than the left, the impulse has a shorter distance to travel and emerges upon the epicardial surface of the right ventricle earlier than upon the epicardial surface of the left ventricle Coincident with the latter part of septal activation the free wall of the right ventricle close to the septum is activated Furthermore, the thinner apical poles are activated earlier than the thicker bases of the ventricles

Electrical Activation of the Ventricles

It will be helpful at this point to consider certain theoretical aspects of the electrical activation first of a muscle strip, then of the human ventricles If a simple muscle strip (Fig 3) is stimulated at point A an electrical impulse spreads from point A along the strip toward point B The impulse can be regarded as a wave front with positive charges in advance and negative charges in the wake of this advancing wave of depolarization A recording electrode is coupled through a galvanometer to a remote point of negligible potential The galvanometer is so arranged that a positive potential at point A is recorded at the galvanometer as an upward deflection and a negative potential at point A as a downward deflection Since point A is in the negative field of the advancing wave during the passage of the impulse, a downward deflection is recorded at A If a similar arrangement is set up at point B, an upward deflection is recorded

at point B since B is in the positive field of the advancing wave It may be concluded, then, that with such an arrangement an impulse moving *from* a point is recorded at that point as a downward deflection and an impulse moving *toward* a point is recorded at that point as an upward deflection By convention the connections to the galvanometer in human electrocardiography are identical with those given above and the same considerations apply

From present knowledge let us now consider (Fig 4) the potential variations occurring from moment to moment during ventricular activation at two points in relation to the heart, one (point A) in relation to the epicardial surface of the right ventricle and the other (point B) in relation

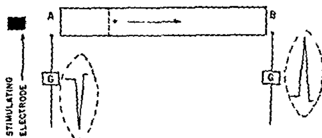


Fig 3 Potential variations at each end of a simple muscle strip stimulated at one end If positivity is indicated by an upward deflection the recording electrode away from which the impulse moves records a downward deflection while that toward which the impulse moves records an upward deflection

to the epicardial surface of the left ventricle An electrode at point A, facing the right ventricle, being in the positive field of the initial unopposed wave moving across the septum, records a small upward deflection (Fig 4, *1*) Additional coalescent wave fronts now form on the left side of the septum and, at the same time, presumably at the apex of the right ventricle near the base of its anterior papillary muscle The electrode is now in relation to a large cone (for although we have represented these electrical events upon the plane of the page they must be thought of in relation to all heart muscle in its three dimensions) of positivity from the spread of the impulse from the left side of the septum, a cone of negativity from the spread of the impulse on the right side of the septum, and a cone of either positivity or negativity from activation of the apical part

of the free wall of the right ventricle depending upon its orientation with regard to the electrode If the cones of positivity predominate there is a further increment (Fig 4, 2) in positivity at electrode A At the next instant (Fig 4, 3) the free walls of the right and left ventricles are being activated The much larger negative fields due to left ventricular activa

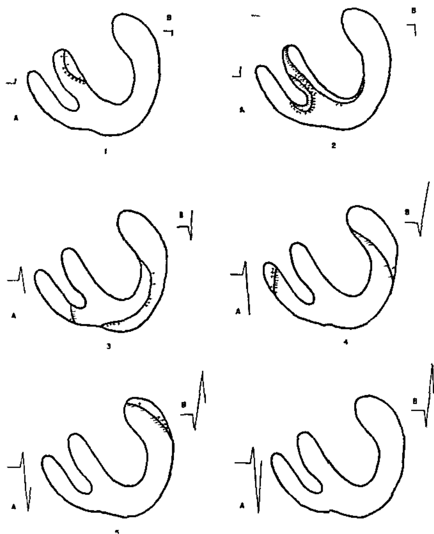


Fig 4 Current conception of the moment to moment manner of depolarization of the ventricles (After Ashman)

tion more than overbalance the small positive field due to right ventricular activation Hence the electrode now shows a change in polarity with consequent reversal in the direction of the deflection recorded there Since the area of negativity facing the electrode becomes still greater the deflection becomes still more negative (Fig 4 4) As the cone of negativity has now reached its maximum and is now decreasing the

deflection has reached its nadir and moves again toward the baseline (Fig 4, 5) Finally with total depolarization of the heart the tracing has returned to the isoelectric line Accordingly the initial ventricular complex as recorded over the right ventricle consists of a small upward R wave followed by a large downward S wave deflection To indicate the relative size of these deflections this may be designated an rS wave These considerations are true whether the electrode is in contact with the right ventricular wall (direct lead) or with the precordium overlying the right ventricle (semidirect lead)

An electrode facing the left ventricle at point B, recording the same electrical events, being in the negative field of the initial unopposed activation of the septum from its left to its right side, records an initial downward deflection At the next instant electrode B is in relation to the larger and nearer field of negativity due to further activation from the left to the right side of the septum, probably overbalancing the smaller and more distant field of positivity emerging from the region of the apex of the right ventricle, a further increment in the initial downward deflection may thus be inscribed For the next few moments depolarization of the free wall of the left ventricle brings a much larger area of positivity into relation with the electrode than the area of negativity produced there by depolarization of the smaller and more distant free wall of the right ventricle Hence the deflection now moves upward When the area of positivity reaches its maximum the upward deflection reaches its apex As the area of positivity in relation to the electrode now recedes the deflection moves toward the isoelectric line, and when the ventricle is fully depolarized the deflection has returned to the isoelectric line Thus in a general way an electrode facing the left ventricle records as the initial ventricular complex a small downward deflection (Q wave) followed by a large upward deflection (R wave) To indicate the relative size of these deflections we may designate this as a qR wave With some qualification these considerations apply equally to an electrode applied directly to the left ventricular surface (direct lead) and to one on the overlying precordium (semidirect lead) The more directly the electrode faces the left side of the septum, the more prominent should the initial downward deflection appear Whatever part of the body faces the left side of the septum should show this initial downward deflection followed by a large upward deflection It should be clear, then, that the normal Q wave is produced by septal activation, it is referred to as a 'septal Q wave' and is a normal finding of no pathologic significance

Precordial Leads

Although some of the pioneer studies in electrocardiography were carried out with chest leads, interest in these leads was abandoned early in favor of study of the electrical events reflected in the frontal plane of the body and recorded in the conventional limb leads However, in the course of some twenty years certain limitations in the conventional limb

leads with regard to the diagnosis of anterior myocardial infarction and certain practical and theoretical developments with regard to the recognition of bundle branch block prompted a renewed interest in these chest or precordial leads At first a bipolar technic similar to that used for the limb leads was employed, the polarity was such that relative positivity at the precordium was represented by a downward deflection of the tracing and relative negativity by an upward deflection Later this convention was reversed so that the tracings would be comparable with those obtained in the three conventional leads It was felt or hoped that the potential of the limb with which the precordial point was paired, had an electrical potential which was negligible with reference to the electrical contribution of the precordial point Actual experience, however, showed that the contribution of the limb potential to the chest lead, whether it was a CF (chest left leg) lead, CL (chest left arm) lead CR (chest right arm) lead or CB (chest-back, usually over the right shoulder) lead, was by no means always negligible The reason for this is twofold (1) Although there is a logarithmic falling off of the recorded cardiac potentials from the heart to the more remote portions of the trunk, which functions as a volume conductor, the limbs act rather as linear conductors and there is therefore no further drop in potential from groin to toes or from axillae to finger tips (2) The heart, as will become clearer presently, may be so oriented as to have a considerable electrical effect at the limb paired with the precordial point Only if the heart happens to be so positioned that a mixture of right and left ventricular potentials, mutually extinguishing one another, is reflected to an extremity, would that extremity show a really negligible potential Therefore none of the limbs can be depended upon to be truly indifferent All workers in the field have therefore agreed upon the desirability of an arrangement one of whose poles records the potentials of a precordial point and the other whose potential is zero, this would be a unipolar chest lead Although opinion is still not unanimous on just how such a unipolar chest lead should be recorded most of the objections to the Wilson unipolar chest lead have now been met This consists of two loops, an exploring loop which is attached to the positive pole of the galvanometer, and a central terminal loop which is connected to the negative pole of the galvanometer The central terminal in turn is connected to each of the three extremities, right arm, left arm and left leg, through a 5000 ohm resistance Accepting the validity of the Einthoven triangle hypothesis, to be described more fully below, the potential at the central terminal must be the average of the potentials at the three extremities (Fig 5) If, for example at one instant during the electrical activation of the ventricles the potential at one of these extremities increases that at the other two extremities automatically decreases so that the average at the three extremities and, therefore, the potential at the central terminal does not change Thus a reference point with a non fluctuating potential is obtained Actual measurements of the potential at this central terminal show it to be very close to zero, with minimal and

unimportant fluctuations from moment to moment during the cardia cycle, and to show less fluctuation than any single point in the body n matter how *remote* from the heart

Although at first the potentials of only one and later two precordia points were recorded, the desirability, in certain conditions, of recordin

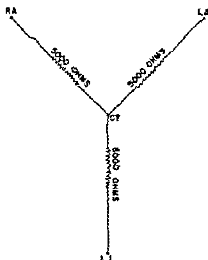


Fig 5 The central terminal of Wilson

Let VR represent the voltage at RA (right arm)
VL represent the voltage at LA (left arm)
VF represent the voltage at LL (left leg)
and VT represent the voltage at CT (central terminal)

But by Ohm s law EMF (voltage in each part of the circuit) = Current (in same part of circuit) × Resistance (in same part of circuit)

Then VR − VT = Current (RA − CT) × a constant
and VL − VT = Current (LA − CT) × a constant
and VF − VT = Current (LL − CT) × a constant

Adding these three equations

VR + VL + VF − 3 VT = [Current (RA − CT) + Current (LA − CT) + Current (LL − CT)] × a constant

But according to Kirchoff s first law the algebraic sum of the currents meeting at any point in any network of wires is zero

VR + VL + VF − 3 VT = 0 × a constant = 0
VR + VL + VF = 3 VT

$$\text{And VT} = \frac{\text{VR} + \text{VL} + \text{VF}}{3}$$

Or stated differently The potential at the central terminal is the average of the potential at the three extremities

the potential at several precordial points is now universally recognized Precordial leads are of value, frequently decisive, in the study of myo cardial infarction, bundle branch block and ventricular hypertrophy In most instances a decision regarding any of these three conditions can be made from the study of two or three precordial points, but there are cer tain instances when decisive changes will be present at only one of six

precordial points This point might be skipped when the potentials of only two or three fixed points are recorded, hence the frequent need for multiple precordial leads Experience has shown that in those cases in which precordial exploration is necessary the potentials of six precordial points should be taken These are

Point 1 At the fourth intercostal space just to the right of the sternum
Point 2 At the fourth intercostal space just to the left of the sternum
Point 3 At a point midway between points 2 and 4
Point 4 At the fifth intercostal space in the midclavicular line
Point 5 At the same horizontal level as point 4 in the anterior axillary line
Point 6 At the same horizontal level as point 4 in the midaxillary line

When these points are paired with the central terminal of Wilson they are called leads V_1, V_2, V_3, V_4 V_5 and V_6 respectively At times it is necessary to record additional leads to the right of V_1 or to the left of V_6 or cephalad to V_4, 5 and 6 This will be discussed later

Extremity Leads

In his classical studies of the electrocardiogram Einthoven conceived of the heart as being in the center of an equilateral triangle whose apices are the right shoulder, left shoulder and left groin, the heart being quite distant from these apices and these apices being distant from one another He assumed also that conduction of an electrical impulse in the tissues of the body from the heart to the extremities is uniform This is the Einthoven hypothesis It is to be distinguished from the Einthoven equation The latter is a necessary consequence of the arrangement of the electrocardiogram and merely states that at any instant the magnitude and sign of the QRS complex (or the P wave or the T wave) in Lead II must be equal to the algebraic sum of the magnitudes of the QRS complex (or P wave or T wave) as inscribed at the same moment in Leads I and III It is a valuable fact often enabling one to detect improper application of the electrodes However there has been some disagreement regarding the validity of the Einthoven hypothesis The points of attachment of the extremities to the trunk do not exactly form an equilateral triangle The heart is not precisely in the center of a triangle formed by the extremities, in general it is closer to the left shoulder than to the right shoulder or left groin Conduction is not uniform through muscle, lung, liver or bone In spite of these objections, however, the hypothesis is close enough to the facts to have been of inestimable value in the study of the electrocardiogram during the past quarter of a century Practically all studies have shown that though there may be some argument about the details in a general way the Einthoven hypothesis may be accepted as valid and workable

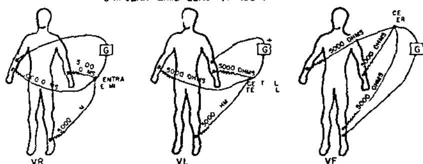
The conventional leads described above being bipolar leads, represent the combined potentials of two of the three points at the apices of the Einthoven triangle Thus a downward deflection in Lead III might be the result of a downward deflection at the left leg or of an upward deflection at the left shoulder, since an upward deflection at the left shoulder

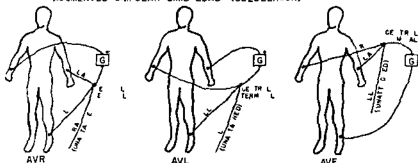
is routed through the galvanometer so that it becomes a downward deflection in writing Lead III As will become apparent, it is frequently desirable to break down the composite potential of Lead III into its constituent parts This can be done by the use of formulas or, much more readily, graphically and strikingly, by the use of unipolar extremity leads These are similar in theory and practice to the unipolar chest leads described above By coupling the central terminal of theoretically zero potential through the galvanometer in turn, to each of the three extremities, right arm, left arm and left leg, it is possible to record virtually the potential at each of these points and thus to obtain, in relatively pure form, as it were, the potential at the apices rather than along the sides of the Einthoven triangle These are called unipolar limb leads and are designated V_R (right arm), V_L (left arm) and V_F (left leg or foot) As worked out by Wilson (Fig 6), a 5000 ohm resistance was included between the central terminal and each of these three extremities to swamp out local differences in resistance between skin and electrode at each of the three limbs In order to increase the magnitude of the unipolar potentials recorded at the limbs Goldberger did two things (1) Contending that little or no difference is produced in the resulting tracings whether or not resistances are included in the circuit, he eliminated the resistances in the portion of the circuit between the central terminal and the electrodes (2) Since the potential at the extremity being investigated was routed to *each* pole of the galvanometer and thus, to a certain extent, was pitted against itself, he detached the wire of the combined portion of the central terminal from the extremity being studied Reference to Figure 6 shows the Goldberger modification more clearly Thus he obtained "augmented" unipolar limb leads, designated aV_R, aV_L and aV_F It is to be noted that, in dropping the central attachment to one of the limbs, the record obtained was no longer strictly unipolar It can be shown, however, that when a similar arrangement is used at each of the extremities this discrepancy is neutralized Wilson accepted the second change but retained the 5000 ohm resistances There is still some controversy as to whether these unipolar limb leads should be recorded at all, whether these unipolar limb leads should be 'augmented' and whether resistors should be retained It is our impression that unipolar limb leads are very helpful in certain cases A study of the recent literature would seem to indicate, moreover, that less error is introduced into the method if the resistors be retained Accordingly the tracings reproduced in this book and labeled aV_R, aV_L and aV_F are Wilson augmented unipolar limb leads

The Electrical Position of the Heart

The heart does not occupy a fixed position in the thoracic cage but may vary in position from individual to individual and from time to time in the same individual It is known that, by and large, the trunk of the body acts as a volume conductor, but the extremities function as *linear* conductors, recording the potentials at their respective points of attach

ment Since these points of attachment are invariable, errors due to slight variations in the point of attachment of the electrode on the limb are obviated, a distinct advantage thus being obtained by the use of the extremities for recording the cardiac potentials In essence then, the





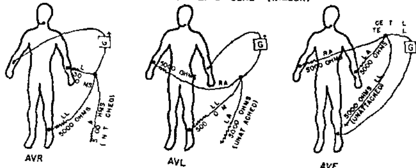


Fig 6 The methods of recording unipolar and augmented unipolar extremity leads

cardiac potentials recorded at a point may vary (1) with variation in the position of the heart in relation to a fixed electrode, (2) with variation in the position of the electrode in relation to a fixed position of the heart, or (3) with a combination of the two By the use of extremity leads the

factor of variation in the position of the electrode is eliminated, enabling us to study variations in the position of the heart

The vantage points used for this purpose are the right arm at its attachment to the right shoulder, the left arm at its attachment to the left shoulder and either the right, or, as is the custom, the left leg at its attachment to the trunk Because of its pericardial reflections the heart is relatively fixed at its base but enjoys a greater degree of mobility at its apex The right shoulder faces the base and cavitary aspect of the heart and hence records a predominantly downward deflection (which may be preceded or followed by a small upward deflection depending respectively upon whether a small part of the right or left ventricular potentials are reflected there) The potentials of the left shoulder and leg, on the other hand, show much greater variations From the latter one may draw certain inferences regarding the electrical position of the heart Assuming normal activation of the heart we may designate as an *intermediate position* (Fig 7), one in which the left ventricular potentials are transmitted approximately equally to the left shoulder and left leg A *horizontal position* (Fig 8) is one in which the heart has rotated on its anteroposterior axis so that the left ventricular potentials are transmitted to the left shoulder while right ventricular potentials are reflected to the left leg A *vertical position* (Fig 9) is one in which the heart has rotated in the opposite direction on its anteroposterior axis so that left ventricular potentials are transmitted to the left leg while right ventricular potentials are reflected to the left shoulder One can, with Wilson, conceive also of semivertical or semihorizontal positions In the *semihorizontal position* (Fig 10) the rotation of the heart is incomplete so that whereas left ventricular potentials are transmitted to the left shoulder, the heart has not developed the extreme degree of rotation characteristic of the truly horizontal heart, and a mixture of right and left ventricular potentials are reflected to the left leg, virtually extinguishing one another, so that the QRS complex recorded at the left leg possesses a low electromotive force In the *semivertical position* (Fig 11), whereas left ventricular potentials are transmitted to the left leg the left shoulder similarly receives a mixture of left and right ventricular potentials, again mutually extinguishing one another, and resulting in a QRS complex of low electromotive force at the left shoulder Comparison of the unipolar limb and chest leads in the following manner enables one to identify the various positions of the heart

- Vertical position Lead V_L resembles Leads V_1 and V_2 Lead V_F resembles Leads V_5 and V_6
- Semivertical position Lead V_F resembles Leads V_5 and V_6 QRS complex of Lead V_L is small
- Intermediate position Leads V_L and V_F are similar and resemble Leads V_5 and V_6
- Semihorizontal position Lead V_L resembles Leads V_5 and V_6 QRS complex of Lead V_F is small
- Horizontal position Lead V_L resembles Leads V_5 and V_6 Lead V_F resembles Leads V_1 and V_2
- Indeterminate position No relation demonstrable between the unipolar limb and precordial leads

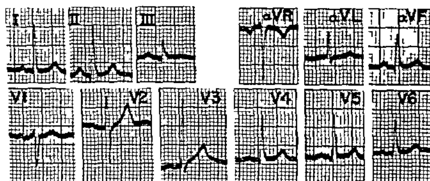


Fig 7 Normal electrocardiogram Heart in intermediate electrical position The left ventricle is so situated that its potentials are reflected equally to the left shoulder and left leg Hence both Lead aV_L and aV_F resemble Leads V_5 and V_6 *It is to be emphasized that although anatomic sketches are here used to illustrate the various positions of the heart this is merely a graphic method of demonstrating how rotation of the heart can alter its electrical projections with regard to the extremities These should therefore be conceived of as electrical positions which may or may not correspond to anatomic positions*

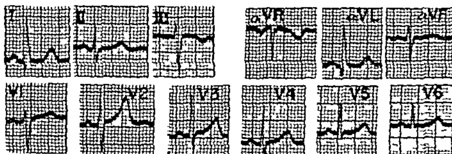


Fig 8 Normal electrocardiogram Heart in horizontal electrical position The heart has rotated in a counterclockwise direction on its anteroposterior and longitudinal axes This swings the left ventricle into relation with the left shoulder and the right ventricle through the diaphragm into relation with the left leg Hence Lead aV_L resembles Leads V_5 and V_6 and Lead aV_F resembles Lead V_1 Left axis deviation here is a positional change and not due to left ventricular hypertrophy The transitional zone lies between Leads V_1 and V_2

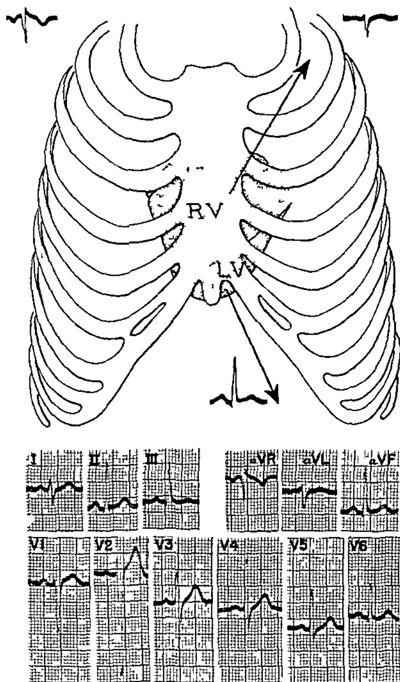


Fig 9 Normal electrocardiogram Heart in vertical position The heart has rotated in a clockwise direction on its anteroposterior and longitudinal axes This swings the right ventricle into relation with the left shoulder and the left ventricle through the left leaf of the diaphragm into relation with the left leg Hence Lead aV_L resembles Leads V_1 and V_2 while Lead aV_F resembles Leads V_5 and V_6 Right axis deviation here is a positional change and not due to right ventricular hypertrophy The transitional zone is at Lead V

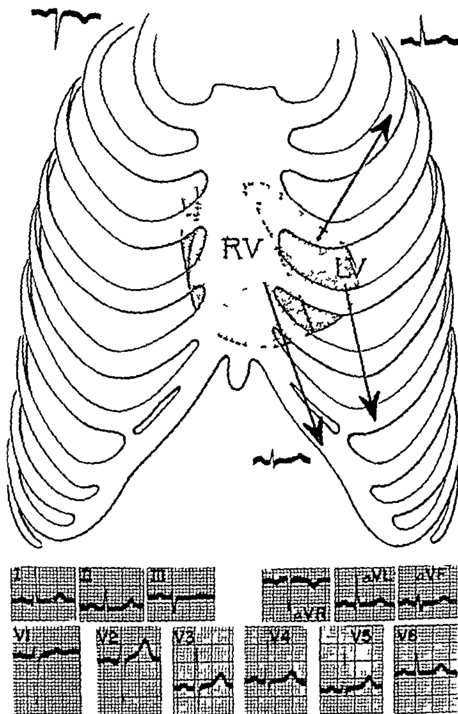


Fig 10 Normal electrocardiogram Heart in semihorizontal position The heart has rotated so that the left ventricle is still in clo e relationship to the left shoulder but counterclockwise rotation of the right ventricle is incomplete so that the left ventricle still has some electrical effect upon the left leg The left leg receives a mixture of left and right ventricular potentials with decrease in the resultant left leg potentials Hence, while Lead aV_L resembles Leads V_5 and V_6 the QRS complex in Lead aV_F has a low voltage

RV

LV

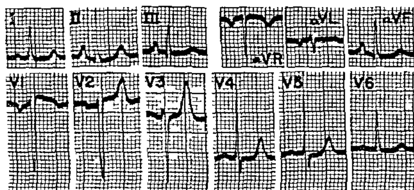


Fig 11 Normal electrocardiogram Heart in semivertical electrical position The heart has rotated so that the left ventricle is still in relation to the left leg but clockwise rotation of the right ventricle is incomplete so that the left shoulder receives a mixture of right and left ventricular potentials with resultant decrease in left shoulder potentials Hence while Lead aV_F still resembles Leads V_5 and V_6 the QRS complex in Lead aV_L has a low voltage The transitional zone lies between Leads V_3 and V_4 Many combinations of axial rotations are possible in the examples given here clockwise rotation on an anteroposterior axis has been combined with clockwise rotation on the longitudinal axis (as viewed from the apex of the heart) and counterclockwise rotation upon the anteroposterior axis has been combined with counterclockwise rotation upon the longitudinal axis

Although roentgen examination usually shows that the anatomic position of the heart corresponds to the electrical position of the heart determined in this way, there may be some exceptions to this Provided these positions be regarded as electrical, these discrepancies need not concern us here The important point is that attention to the electrical position of the heart often enables us to detect from the electrocardiogram changes in its appearance which are referable, not to disease of the heart, but to changes in its electrical position

The Conventional Limb Leads

Einthoven, impressed with the relatively fixed position of the extremities with relation to the heart, focused the attention of workers in the field upon the use of the limb leads derived from the three points of the triangle formed by the right and left shoulder and the left leg Thus for some twenty-five or thirty years electrocardiography was dominated by Einthoven's three extremity leads Lead I he designated as the potential difference between the right and left shoulder and is recorded by routing the left arm potentials through the string galvanometer so that a positive potential at the left shoulder is written upward, while the right arm potentials are so routed that a positive potential at the right arm is written downward Thus Lead I is a bipolar lead and the composite result of the potentials at the right and left shoulder, the left shoulder contributing to Lead I with unchanged polarity while the polarity of the right shoulder potentials is reversed in Lead I Similarly Lead II records the difference in potential betwen the right arm and left leg recorded simultaneously, the right arm potentials entering in a negative and the left leg potentials in a positive way into the resultant potentials Lead III is the composite effect of the potentials at the left arm and left leg, the left arm potentials being routed to the negative and the left leg potentials to the positive pole of the galvanometer Most of our knowledge of cardiac arrhythmias, conduction disturbances and abnormalities of the auricular and ventricular complexes was worked out with these bipolar limb leads However, the limitation of the method with regard to certain cases of myocardial infarction and bundle branch block later redirected attention to the study of the precordial leads Somewhat later the desirability was appreciated of breaking down the precordial and conventional extremity leads into their constituent parts and thus led to the development of unipolar lead electrocardiography Although it is possible that the use of unipolar limb leads may eventually supplant conventional leads, there are several factors, including current usage of the latter and inadequate experience with the normal variations in the former, which compel the retention of the conventional leads at least for the present Accordingly a description of the normal variations in the three conventional limb leads is warranted

When the electrocardiogram of a normal individual is taken a series of waves is recorded They have been arbitrarily called P, Q R, S and T waves (Figs 12, 13, 14, 15) These electrical complexes differ in form in the various leads The P wave represents the electrical disturbances that

take place in the auricles The first half or so of the P wave represents right auricular activation, the second half left auricular activation Because of the situation of the auriculoventricular node in the inferior portion of the right atrium, the impulse enters the auriculoventricular node at or shortly before the peak of the P wave The P wave as well as the other important waves are apt to be most prominent in Lead II All of the other waves are due to ventricular activity and can be divided into two

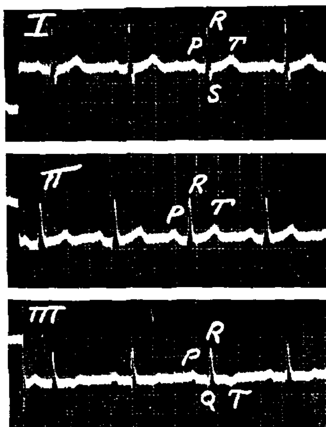


Fig 12 Normal mechanism The three curves show the three conventional leads At the beginning of each tracing the standardization is photographed (1 centimeter = 1 millivolt) P represents auricular depolarization QRS is the initial (depolarization) and T the final (repolarization) ventricular complex The time is indicated in 1/25 and 1/5 seconds Note that T_3 may be flat normally

portions the initial deflections (QRS) and the terminal deflection (T) The first downward wave, if followed by an upward deflection, is called a Q wave The first upward wave whether preceded by a downward one or not, is called an R wave A wave that is directed downward which follows an R or is not followed by an R is called S If there is a second upward deflection after an S wave, it is called R and similarly a second downward deflection after R would be S If there is only one deflection

and it is downward it may be called QS There is some controversy about the proper terminology of the initial deflections but the above nomenclature has the sanction of present usage After the QRS complex there follows a brief isoelectric line which gradually blends into a smooth

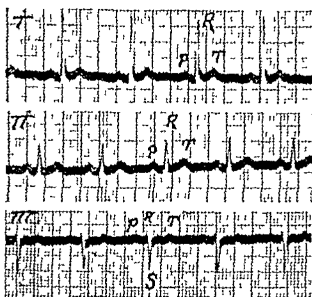


Fig 13 Normal curves This patient aged 49 felt well and showed nothing unusual on physical examination and yet seven days later had a typical coronary thrombosis from which he recovered

rounded T wave Occasionally there may be seen a small U wave after the T which has an obscure origin and no practical significance

The P wave normally is upright and measures about 0.5 to 2.5 mm in height The P Q or P-R interval which measures the time it takes an impulse to go from auricles to ventricles (conduction time) varies from 0.12 to 0.20 second Beyond this it is regarded as pathologic Most of

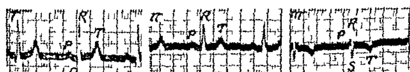


Fig 14 Normal curves Note the inverted T_3 and the slight tendency to left axis shift which are common findings in normal persons, especially after the age of 40 and in stocky individuals

this delay takes place at the auriculoventricular node and bundle of His The normal P R interval decreases with increasing heart rates and is shorter in children than in adults In children under 6 years the range is 0.13 to 0.17 seconds and for those between 7 and 13 years it is 0.14 to 0.18 seconds for rates of 130 to 70 respectively Normally Q and S waves may or may not be present When the heart is rotated on its longitudinal

axis in a counterclockwise direction as viewed from the apex, a Q wave is present in Lead I and an S wave in Lead III On the other hand, when the heart is rotated on its longitudinal axis in a clockwise direction as viewed from the apex, the S wave is present in Lead I, the Q wave in Lead III The R wave varies in height from about 5 to 15 mm The T normally is upright in Leads I and II and may be upright or inverted in

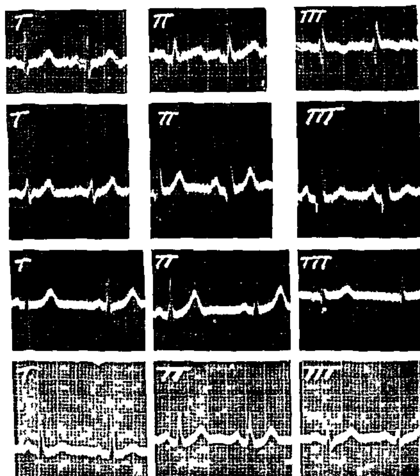


Fig 15 Four sets of normal curves The first is from a child 8 years of age The next three are from adults 23 40 and 53 years of age None had any evidence of heart disease Note variations in the form of the normal electrocardiogram

Lead III Its amplitude will range from 1 or 2 mm to 4 or 5 mm The duration of the QRS is an important part of the study of a tracing Normally it will be found to be between 0 04 and 0 08 second When it reaches 0 1 second it is regarded as delayed The duration of the Q T interval which is an accurate measurement of the length of ventricular electrical systole is about 0 4 second but will vary with the cardiac rate

The QRS complex really measures the time it takes the impulse

spread throughout the two ventricles and reflects the integrity of the two branches of the bundle of His The thickness of ventricular muscle will affect this time to only a slight extent The T wave or terminal portion of the ventricular complex may be regarded as due to the process opposite to that producing the QRS The latter is the advance of the electrical or physicochemical process, the former is the retreat

Normally the P, R and T waves which are regarded as the more constant deflections should be greatest in Lead II and the sum of the height of the waves in Leads I and III should equal Lead II Because of the great variations in the character of these complexes in normal individuals, a considerable experience is required to become familiar with the normal and caution must be exercised before slight alterations are regarded as significant

Recently it has been suggested that during the last two months of pregnancy electrocardiographic evidence of the fetal heart can be obtained by taking a lead from the left arm and right leg of the mother Very minute waves may be seen regularly interspersed among the mother s electrocardiographic tracings Insertion of electrodes into the rectum or vagina of the pregnant woman may enable one to obtain larger electrocardiograms of the fetal heart

THE NORMAL PRECORDIAL ELECTROCARDIOGRAM

We have seen that it does not matter whether an electrode is applied an inch higher or lower on one of the limbs since the limbs function as linear conductors However, in taking leads from the chest, moving the electrode 1 inch over the precordium produces profound alterations in the tracings This introduces some confusion in comparing precordial tracings obtained in the same patient at different times The position of the heart may change with relation to the six designated precordial points Furthermore, the precordial electrode may not be applied to exactly the same point on the chest wall at each recording By taking the entire six precordial leads it is possible, often at a glance, to eliminate changes which are due to either of these changes, i e , change in the position of the heart itself, or a change in the position of the electrode Herein lies one of the most important reasons for recording six rather than one, two or three precordial leads

It is to be remembered that each precordial lead records the potential not only of the part of the heart immediately underneath the electrode but also, to varying degrees, that of more remote parts of the heart If the heart is close to the chest wall the effect of the adjacent part of the heart upon the electrode will be relatively greater than the effect upon this electrode of more remote parts of the heart Conversely, if the heart is deep in the chest and relatively distant from the precordium there will be a greater relative effect of the more remote portions of the heart upon the electrode In the latter instance the changes from V_1 to V_6 will proceed more gradually because at each precordial position a fusion of the potential of many parts of the heart is produced On the other hand, when

the heart lies closer to the chest wall abrupt changes may be detected from one position to the next Ordinarily the spectrum of changes consists of a progressive change from right to left ventricular potentials, however, if the heart happens to show extreme rotation Lead V_1 need not show characteristic right ventricular potentials or Lead V_6 need not show characteristic left ventricular potentials For example, if the heart rotates clockwise on its longitudinal axis (as viewed from the apex) and the apex is also displaced posteriorly Lead V_6 may still show the rS pattern characteristic of right ventricular potentials (Fig 167) In that case it would be necessary to take additional leads to the left of Lead V_6 at V_7 (same horizontal level in the posterior axillary line) or at V_8 (same horizontal level over the scapula), or cephalad to these positions in the third or fourth interspaces in order to record true left ventricular potentials On the other hand, the heart may be rotated counterclockwise on its longitudinal axis so that Lead V_1 records left rather than right ventricular potentials In such a case (Fig 26) it has been recent practice to take an additional lead to the right of Lead V_1 at a position having a similar relation to the right anterior chest to that which Lead V_3 has with relation to the left anterior chest This is designated V_{3R} or V_3

Ordinarily Lead V_1 may or may not show an R wave Absence of an R wave at this location may therefore be a normal variation, and may be assumed to result from the relationship of the electrode to the right ventricular cavity with septal activation proceeding tangentially with relation to the electrode When an R wave is present it is probably due mainly to septal activation The S wave is generally quite large The T wave at this location may be inverted, isoelectric or upright

At position V_2 an R wave is usually recorded If there was an R wave at V_1, it is slightly taller at V_2 than at V_1 The greater size of the R wave is probably related to the closer relationship of the electrode with the septum and the fact that it now faces the septum more directly SV_2 is as a rule somewhat deeper than SV_1, this is probably due to closer proximity of V_2 to the heart in most cases If the heart were strongly rotated counterclockwise on its longitudinal axis SV_1 would be larger than SV_2 because V_2 would be closer to the left ventricle and to the larger R type of potential The T wave in adults is generally upright from V_2 to V_6 In children, young women and as has been reported in Negroes, the T wave may be inverted in V_2, V_3 or even V_4 The exact frequency of this finding in the normal adult population has not yet been established

The R wave in Lead V_3 is still taller, by the time this point has been reached it generally measures 2 mm or more Failure of the R wave to increase as the electrode moves toward the left is an abnormal finding This may result from anteroseptal scarring from an old myocardial infarct with resultant failure of the subjacent muscle to be activated Even more suggestive of scarring would be a decrease in the magnitude of R once it has appeared at Lead V_1 or V_2 In other words the R wave should normally show a progressive increase from Lead V_1 to V_4 If it fails to increase or, more strikingly, if it decreases, there may be an infarct of the

myocardium in this region This change may also occur with left ventricular enlargement, particularly if associated with incomplete left bundle branch block The R wave fails to increase in left bundle branch block because in that disturbance the septum is activated from its right to its left side The wave which would proceed early from left to right fails to appear and thus fails to antagonize the downward impulse recorded at the right precordium corresponding to activation of the septum from its right side

Coincident with increase in the height of the R wave in Lead V_3 the S wave decreases In some cases, especially when the heart is in a vertical position, RV_3 may actually be larger than SV_3, but there is considerable variation from heart to heart in the point at which R first exceeds S

Usually at Lead V_4 the R wave is much taller and the S wave much smaller The R wave here is due to activation of the anterior or anterolateral wall of the left ventricle If the apex is well anterior the S wave may now be quite small or even absent If the apex is displaced posteriorly the R wave may be smaller and the S wave still relatively prominent The presence of an S wave signifies that more of the left ventricle (its posterolateral aspect normally) is activated after the muscle near the electrode If no S wave is inscribed one may conclude that the electrode is in relation to the last part of the left ventricle to be activated

Because of the intervention of relatively poorly conducting lung tissue between the lateral aspect of the heart and the precordium the magnitude of all deflections may fall off in Leads V_5 and V_6 In fact, miniature tracings maintaining the same type of relationship between R and S are not at all uncommon in Lead V_6 In many cases, especially if the heart is in a horizontal position, the R and T waves may be as large in V_6 as in V_4 or even taller The important point, however, is that whereas a decrease in the height of the R wave from Lead V_1 to V_4 is abnormal, a falling off of the R wave from Lead V_4 to V_6 need not be

A Q wave is not normally seen in Leads V_1 to V_3 As the electrode moves to the left and faces more and more directly into the left side of the septum, a Q wave appears and becomes more prominent in relation to the R wave The exact point at which this Q wave is first encountered in going toward the left of the precordium varies, of course, with the position of the septum It generally does not become well marked until Lead V_5 or V_6 has been reached, and may not be recorded at all in the usual six chest leads This Q wave is thin, rarely lasting longer than 0 02 second, and, even when most prominent in comparison with the R wave in the same lead, never measures more than a small fraction of that R wave This Q wave, as we have seen, is a normal finding due to septal activation and has no pathologic significance

The "Intrinsicoid Deflection"

If a third recording electrode is placed about halfway along the experimental muscle strip shown in Figure 3 a biphasic (±) deflection is there

recorded as the wave of excitation travels along the muscle strip This is due to the fact that this new point functions in two ways, first as electrode B or a point toward which the impulse travels, then as electrode A or a point away from which the impulse moves Hence an initial upward and a final downward deflection are inscribed The moment when the impulse has arrived at or near the muscle beneath the electrode is signalled by the sudden change in the direction of the impulse and is customarily measured at the beginning of this intrinsic deflection Although for some time a similar terminology has been used with regard to points over the precordium, it should be clear that in this case the electrodes and the muscle being activated are relatively remote from one another The final downward deflection there has less magnitude and may appear less rapid than directly over the epicardium Furthermore, the electrode is not arranged parallel to the line of activation of the ventricles Rather, in a general way, the impulse, moving from endocardium to epicardium, approaches the electrode, then emerges upon the epicardial surface of the ventricles In terms of the bipole theory the beginning of the final rapid downward deflection coincides with the moment when the largest field of positivity is oriented toward the electrode As the field of positivity begins to decrease the deflection recedes toward the isoelectric line This does not necessarily mean that the impulse emerges on the epicardium beneath the electrode at that moment Actually this is more apt to occur some time on the downstroke of the rapid deflection The term 'intrinsic deflection, then, is a concept useful in the experimental study of muscle activation while the term intrinsicoid deflection has been proposed as a more appropriate term for the clinical appraisal of electrical activation of portions of the intact heart

Whatever its mechanism, the measurement of the time of this deflection over the precordium has proved of great value in the study of the normal or hypertrophied heart or the heart with bundle branch block As a matter of convenience this may be measured from the beginning of the QRS complex to the apex or starting-off point of the rapid downward deflection (ordinarily the beginning of the RS wave), if there is more than one rapid downward deflection the final one should be taken as the intrinsicoid deflection To be quite accurate this deflection would have to be measured from the beginning of the earliest Q wave in a simultaneously recorded conventional limb lead, however, since simultaneous leads are not taken in ordinary clinical electrocardiography the deflection is measured as accurately as possible, from the beginning of the QRS complex in the same lead

Because the right ventricle is normally thinner than the left, the impulse activating its wall has a shorter distance to travel than has an impulse activating the left ventricle Accordingly the intrinsicoid deflection, which we have seen gives a measure of the time of activation of the ventricle, is inscribed earlier over the right than over the left ventricle Ordinarily the difference between right and left ventricular activation does not exceed

0 02 to 0 025 second In left ventricular hypertrophy the difference may approach or exceed 0 045 second This delay parallels the increase in the voltage of the R wave over the left ventricle but it is to be noted that ventricular thickness is not the only factor affecting the voltage of the R wave The proximity of the ventricle to the chest wall and probably certain chemical changes at the boundary of the heart muscle cells are two other factors to be considered In right ventricular hypertrophy, apparently depending upon its degree, the difference between the intrinsicoid deflection over the right and left ventricles may be decreased or, perhaps more often, the intrinsicoid deflection may be inscribed later over the right than over the left ventricle *In left bundle branch block the delay* in activation of the left ventricle is extreme In complete or incomplete right bundle branch block the reversal in the normal relationships of right and left ventricular activation is invariable

The "Transitional Zone"

The point or area over the precordium where the form of the ventricular complex changes from that characteristic of the right ventricular potentials in that heart to that characteristic of left ventricular potentials in that same heart is referred to as the transitional zone " In a general way this indicates the position of the interventricular septum If the heart lies close to the chest wall the transition may be an abrupt one, changing from a right to a left ventricular potential from one of the usual precordial points to the next If, on the other hand, the heart lies deep in the chest, the change may be smoother and slower, showing a gradual evolution from right to left ventricular potentials over two, three or more precordial points The change is more readily determined if the direction of the projection of the interventricular septum upon the precordium is at right angles to the line of the usual six precordial points, and less readily determined if the projection of the septum and this line are parallel to one another *The change may be from the rS complex of the right ven* tricle to the qR complex of the left ventricle Very often, however, notching of the QRS complex is noted at the transitional area, an initial R wave corresponding in time to the R wave of one ventricle, and a secondary R wave to that of the other ventricle (Fig 26) This type of notching *is a normal variant and is not to be confused with that due to com*plete or incomplete bundle branch block or to focal block One cannot be sure of block of either of these types unless the electrode lies clearly in relationship either with the right or the left ventricle In deciding whether one has recorded true right and left ventricular potentials, it is essential to demonstrate that the "transitional zone has been crossed, either with the use of the usual six precordial leads, or, if necessary, with additional leads to the right or left of these As a corollary to this fact, the position of the 'transitional zone is of some value in determining rotation of the heart on its own longitudinal axis A pronounced shift of the transitional zone to the left may be an important clue to, and

in rare cases the sole electrocardiographic evidence of, acute cor pulmonale When the transitional zone is in relationship with either the left shoulder (aV_L) or the left leg (aV_F), bizarre notched low voltage complexes are apt to be reflected to either location This constitutes one of the criteria for the designation of a semivertical heart in the former instance or of a semihorizontal heart in the latter If the voltages are small (less than 3 or 4 mm) at either of these points, analysis of the relative duration and amplitude of the individual components of these deflections is likely to prove misleading in the study of myocardial infarction Furthermore, one must distinguish between the transitional zone demarcating left and right ventricular potentials from that demarcating infarcted and uninfarcted muscle This problem is more fully considered in the discussion of myocardial infarction

VENTRICULAR HYPERTROPHY

The height of the complexes in the three conventional leads is determined in part by the electrical axis of the heart Ordinarily R waves are highest in Lead II and the sum of the R waves in Leads I and III is approximately equal to that in Lead II Since the three conventional leads represent the sides of the Einthoven triangle the magnitude of the QRS complex in two of the three conventional leads represent the projection of a mean electrical axis of the heart upon these sides of the Einthoven triangle From the magnitude of these deflections we can calculate the direction and magnitude of the mean electrical axis which must produce them We arrive then at a vector quantity which has magnitude and direction Although authorities have set somewhat different limits to the range of normality for this vector, most observers agree that the range from 0 to 90 degrees should be considered normal (Fig 16) All hearts with axes to the left of 0 degrees or thereabout are considered to show left axis deviation and all those to the right of 90 degrees to show right axis deviation (Fig 17) Generally when the highest upward deflection (R wave) occurs in Lead I and the lowest downward deflection (S wave) in Lead III it denotes left axis deviation If the changes are reversed and the lowest downward wave is in Lead I and the highest upward deflection is in Lead III right axis deviation is indicated In a majority of cases leftward deviation of the axis is associated with left ventricular hypertrophy and rightward deviation of the axis with right ventricular hypertrophy However as was appreciated early in the development of electrocardiography by Einthoven himself, the electrical axis might lie outside of the so-called normal range in the absence of organic heart disease as a result of abnormal positions of the heart being pushed to either one side of the chest or another by fluid in the pleura by a high diaphragm or by a malformation of the thoracic cage In rare cases left ventricular hypertrophy has even been found at postmortem examination in individuals whose electrocardiograms showed right axis deviation during life Left axis deviation moreover can often be made to disappear by

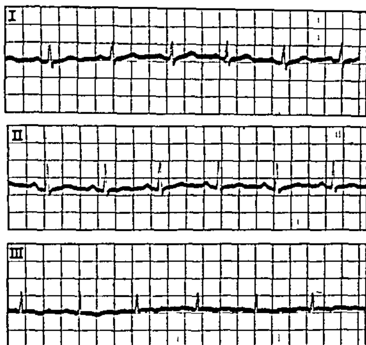
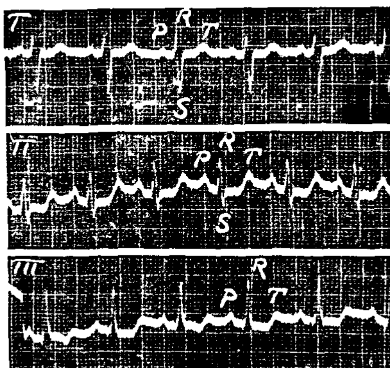


Fig 16 Normal electrical axis The R wave in Lead I measures 4 millimeters the S wave 1 millimeter The net deflection in Lead I is plus 3 millimeters The R wave in Lead III measures 4 5 millimeters Perpendiculars dropped from the positive side of the Lead I side of the Einthoven triangle and from the positive side of the Lead III side of the Einthoven triangle meet at the tip of the arrow This points within the normal range of electrical axes (zero to plus 90 degrees)



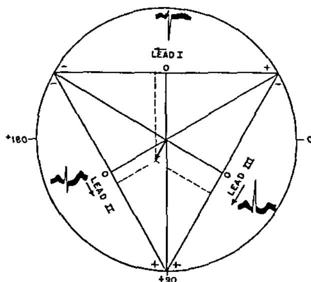


FIG 17 Right axis deviation Note that the highest upward wave (R) of the initial ventricular deflection is in Lead III and the lowest downward wave (S) is in Lead I The projection of the QRS complex is on the negative half of the Lead I side of the Einthoven triangle and on the positive half of the Lead III side Perpendiculars to these projections intersect in the left lower quadrant of the triangle between plus 90 and plus 180 degrees This corresponds to right axis deviation. This patient had mitral stenosis

having the patient take a deep breath (Fig 18) With the descent of the diaphragm the deep S_3 may diminish in size and even give way to an upright R_3 It may be concluded, then, that a statement regarding the mean electrical axis must be considered inconclusive evidence for ventricular hypertrophy *Therefore, since the mean electrical axis is not decisive its calculation is not recommended as a routine in clinical electrocardiography*

By the use of unipolar extremity and chest leads, however, it is possible readily to separate the effects of the position of the heart from those due to ventricular hypertrophy In most cases the electrical position of the heart can easily be determined by comparison of the potentials at the left shoulder and leg with those at V_1 and V_2 and at V_5 and V_6 in the manner previously described In general, hearts in a horizontal or semihorizontal

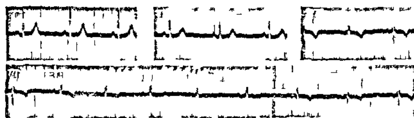


Fig 18 Effect of deep breath on left axis deviation The patient was a woman 45 years old with a normal heart Note disappearance of S_3 and upright T_3 with deep inspiration

position show left axis deviation and hearts in a vertical or semivertical position show right axis deviation

Left Ventricular Hypertrophy

In classical left ventricular hypertrophy the R waves are characteristically very tall over the left ventricle (usually in Leads V_5 and V_6), a small, narrow Q wave is present in one or more of these leads, especially in one facing directly upon the left side of the septum, the RS-T segment is depressed and the T wave inverted in all or some of the leads in which tall R waves are inscribed (Figs 19 to 23) As pointed out above it may be necessary to record additional leads to the left of Lead V_6 in order to record true left ventricular potentials Corresponding to these changes over the left ventricle smaller R waves, deeper S waves, reciprocal elevation of the RS-T segment and upright T waves may develop over the right ventricle (Leads V_1 and V_2), but these changes are not always present

If the left ventricle is in relation to the left shoulder similar potentials will be manifested at Lead aV_L and, since Lead aV_L contributes to Lead I in a positive way, in Lead I as well In fact at times, when true left ventricular potentials are not transmitted to the usual precordial points, the diagnosis of left ventricular hypertrophy may be made on the basis of these changes at the left shoulder The duration of the QRS complex, which should be measured in the conventional leads, is apt to be increased

to from 0 09 to 0 11 second When this measures or exceeds 0 12 second it is more likely to be due to a delay or block in one of the branches of

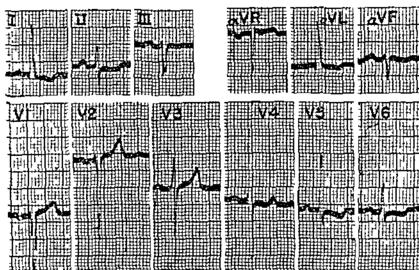


Fig 19 Left ventricular hypertrophy Heart in horizontal electrical position Left axis deviation Tall R waves in Leads V_4 and V_5 depressed RS T segments and inverted T waves in Leads V_5 and V_6 indicate left ventricular hypertrophy Since Lead aV_L resembles Leads V_5 and V_6 while Lead aV_F resembles Leads V_1 and V_2 the heart is in the horizontal electrical position

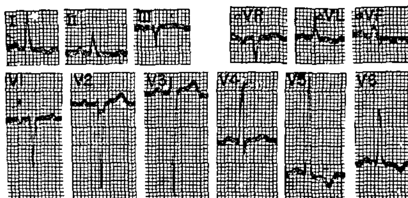


Fig 20 Left ventricular hypertrophy Heart in semihorizontal electrical position The deep S waves in Leads V_2 and V_3 tall R waves in Leads $V_{4\ 6}$ and depressed RS T segments and inverted T waves in Leads V_5 and V_6 establish the diagnosis of left ventricular hypertrophy The intrinsicoid deflection in Lead V_6 is measured at 0 04 second The ventricular complex in Lead aV_L resembles those in Leads V_5 and V_6 while QRS V_F is of low voltage indicating a semi horizontal electrical position of the heart

the bundle of His Corresponding to the slight prolongation of the QRS complex there is a greater than normal delay in the timing of the intrinsicoid deflection over the left ventricle, the latter being inscribed 0 04 to

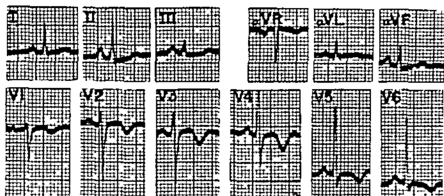


Fig 21 Left ventricular hypertrophy Heart in intermediate electrical position Superimposed T wave changes The patient was a 63 year old woman with long standing hypertension who was admitted because of recent angina pectoris The tall R waves and inverted T waves over the left ventricle indicate left ventricular hypertrophy The QRS complexes in Leads aV_L and aV_F resemble those in Leads V_5 and V_6 The heart is therefore in the intermediate electrical position With left ventricular hypertrophy one would expect upright T waves in Leads V_{1-3} reciprocal to the inverted T waves in Leads V_{4-6} The fact that $TV_{1\ 3}$ is inverted instead suggested anterior myocardial ischemia or infarction Subsequent electrocardiographic observation (see Fig 158) showed waxing of these T wave changes confirming this impression

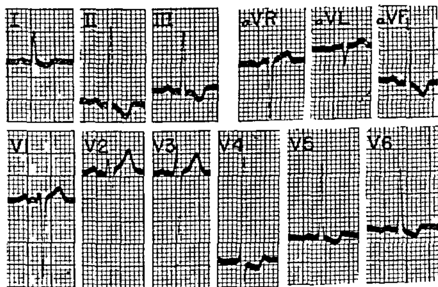


Fig 22 Left ventricular hypertrophy Heart in semivertical electrical position The electrical axis is normal Tall R waves, depressed RS T segments and inverted T waves in Leads V_{4-6} indicate left ventricular hypertrophy Note reciprocal elevation of RS T segments in Leads V_{1-3} a common accompanying finding not diagnostic of anterior myocardial infarction Lead aV_F resembles Leads V_{4-6} while Lead aV_L shows a low voltage QRS complex This places the heart in a semivertical electrical position Unless the precordial leads were examined one could readily mistake this for right ventricular hypertrophy

0 05 second later over the left than over the right ventricle The increased height and delayed peak of the R wave are due to the greater thickness of the left ventricle the impulse reaching the surface of the left ventricle after a greater than normal delay It must be remembered, however, as mentioned above, that the voltage of the R wave is a function of other factors than ventricular thickness The proximity of the heart to the precordial electrode, certain chemical changes such as the serum potassium level, and perhaps the state of cardiac compensation also have their effect upon R wave voltage

Recent experience has shown that the diagnosis of left ventricular

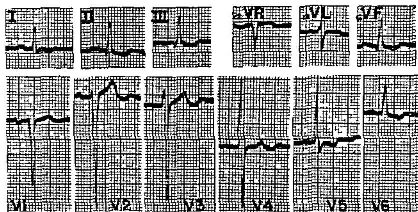


Fig 23 Left ventricular hypertrophy Heart in vertical electrical position Normal electrical axis The totality of changes in Leads V_{4-6} namely tall R waves in Leads V_4 and V_5 depressed RS T segments and inverted T waves in Leads V_5 and V_6 indicate left ventricular hypertrophy Note that deep S waves elevated RS T segments and upright T waves in Leads V_{1-3} mirror the changes in the left ventricle In doubtful cases many authorities take these changes into con sideration in making the diagnosis of left ventricular hypertrophy It should be remembered that depth of S waves or height of R waves may be functions of the proximity of the heart to the precordium as well as of ventricular thickness The identity of Lead aV_F with Lead V_6 and the resemblance of Lead aV_L with Lead V_1 would place this heart in a vertical electrical position If the precordial leads were neglected one might erroneously consider this right ventricular hypertrophy

hypertrophy made upon the basis of the above enumerated criteria was invariably confirmed at postmortem examination, but the additional pres ence of right ventricular hypertrophy or old myocardial infarction was usually missed electrocardiographically When the autopsy material was considered from the opposite point of view, examining the electrocardiographic findings of all individuals showing left ventricular hypertrophy at autopsy, it was found that two thirds of them showed characteristic electrocardiographic changes of the type described above Abnormal nonspecific changes were recorded in the electrocardiograms of the remaining one third but a few showed bundle branch block and in rare instances normal electrocardiograms were recorded In an attempt to make a correct

anatomic diagnosis in some of the cases that would otherwise be missed the inclination in many quarters has been to broaden the criteria upon which the diagnosis may be hazarded Thus some authorities feel that if the QRS complex measures 0 09 second in duration, if the R waves are exceptionally tall and wide in some of the conventional leads or in Leads V_4 through V_6, if the S waves are exceptionally wide and deep over the right ventricle or in all of the conventional leads, even if accompanying RS-T segment and T wave changes are absent, or if certain prescribed combinations of these findings are present, the diagnosis of left ventricular hypertrophy may be ventured However, experience has shown that while the number of positive diagnoses is thus increased, the number of false positives is also increased A statement that these secondary criteria are *suggestive of or consistent with left ventricular hypertrophy would* perhaps be the wiser policy

The Ventricular Gradient, Primary and Secondary T Wave Changes

At this point a theoretical digression into the concept of the ventricular gradient will be most helpful If an ideal cell or muscle strip of the type described in Figure 3, possessing uniform physiologic properties, is stimulated, a wave of depolarization sweeps over this cell or muscle strip from the point of stimulation If an electrode toward which this impulse moves is connected to the positive pole of a galvanometer and through the galvanometer to another electrode of negligible potential, the movement of the impulse toward the electrode will be recorded at the galvanometer as an upward deflection This deflection has *large magnitude* and *short duration* The identity of this deflection with the QRS complex of the electrocardiogram will be apparent Now after a pause the local changes in permeability at the surface of the cell or fiber tend to disappear, the original dielectric effect at the surface is then restored in the same order in which it disappeared This change occurs much more slowly and the magnitude of the changes reappearing at the cell from moment to moment is much smaller than during the original process of depolarization The restoration process is referred to as repolarization Since the electrical changes at the cell surface are now opposite in direction to those which developed during electrical activation of the cell or fiber, the deflection recorded at the galvanometer during this restitution process will have an opposite *direction to those initially* inscribed The repolarization process ideally then is one of *lesser magnitude, greater duration and opposite sign* to the depolarization process Its resemblance to an inverted T wave of the electrocardiogram will be obvious Since the cell is assumed to be of uniform physiologic properties the area enclosed above this wave must equal that enclosed under the R wave If the area above this T wave does not exactly equal that under the R wave or, in other words, if the sum total of the area under the QRS complex and that above the T wave (due regard being had for the signs of these deflections) does not equal

zero, then the cell or fiber is not of uniform physiologic properties This in a nutshell is the concept of the ventricular gradient The existence of a gradient, or difference, indicates local differences in the properties of the cell or fiber These differences can be the result of differences in the process of depolarization or of repolarization The latter is, however, much more easily affected by such physical or chemical changes as local cooling, local anoxia and local intoxication than is the former Only a much more severe grade of injury is capable of altering the depolarization process This corresponds to the clinical fact that the T wave is much more labile and easily altered than is the QRS complex The change in the repolarization process is manifested by a greater slowing or delay in the time of recovery in one part of the cell or fiber than in another Worded differently, there is a local increase in the duration of electrical systole In the ideal cell or fiber described above, the T wave has an opposite direction to the QRS complex Physiologists have long been puzzled by the fact that in the human heart the T wave normally has the same direction as the T wave in Leads I, II and over the left ventricle This must be explained by assuming that in man the physiologic properties of the normal ventricle are not uniform and that there are local differences in the duration of electrical systole In other words, there is normally a gradient in the human heart

It will be obvious on the basis of the ventricular gradient concept that T waves may be considered in relation to the QRS complexes preceding them If the gradient is zero, the area of the T wave may be explained as a necessary consequence of the area of the QRS complex and the process of repolarization is entirely accounted for by the process of depolarization In this case the T wave may be considered secondary to the QRS complex Thus if the ventricle is merely activated in a different direction without changing its physiologic characteristics as in ventricular premature beats or in experimental bundle branch block, secondary T wave changes develop, the gradient is the same in the regular as in the premature or in the aberrant beats If, on the other hand, the area of the T wave cannot be explained as a necessary consequence of the area of the QRS complex a gradient exists and the T wave shows primary changes Primary T wave changes may be seen in myocardial ischemia, as the result of local chilling of the heart by drinking ice water, in inflammatory diseases of the myocardium in trichinosis and in such toxic conditions as diphtheria or emetine poisoning It is the purpose of the determination of the ventricular gradient to separate these primary T wave changes from the less significant secondary T wave changes

No attempt will be made here to describe in detail the method of measuring the ventricular gradient Suffice it to say it consists of measuring the difference of the area of the QRS complex and of the T wave as projected in the frontal plane of the body by appropriate measurements in two of the three conventional leads, preferably recorded simultaneously Until such time as an accurate practical clinical method of determining the

ventricular gradient is available it may be adequate in most cases to judge by inspection and from experience whether the RS-T and T wave changes may or may not be accounted for as consequences secondary to the QRS changes

"Left Ventricular Strain"

We have been impressed with the variability of the electrocardiographic appearance in hospitalized patients with left ventricular hypertrophy On one day the R waves might be tall and the corresponding T waves rather small, on another day there might be characteristic RS-T and T wave changes in addition to tall R waves, on another normal sized R waves with inverted T waves and, on still another, normal complexes Of course this does not imply that anatomic hypertrophy was present at one time and not at another For a variety of reasons, among them this observation that other factors than hypertrophy may affect the resultant electrocardiogram, the designation of "left ventricular strain' has come into rather widespread use, but the exact meaning of the term has varied from one authority to another Some consider ' strain" to represent the response of a ventricle to a stress sometimes chemical, sometimes mechanical and sometimes positional However, there are certain unwarranted implications in the use of such a term It might be considered that the hearts of patients with the so-called "left ventricular strain pattern' are near the breaking point, yet experience has shown that this ' strain pattern ' may persist for twenty or thirty years or even longer Furthermore, as a matter of experience, the progress of electrocardiography has been impeded in the past because mechanical implications have been drawn from electrical changes, witness the confusion over a number of years of the T wave with mechanical diastole It must be conceded that this same criticism can with equal justification be made against the term left ventricular hypertrophy It has even been suggested that left ventricular hypertrophy and left ventricular strain are two different concepts and that, whereas hypertrophy produces only high voltage QRS complexes, strain produces only changes in the RS-T segment and the T waves The validity of this distinction is open to question, but it is possible that this concept may offer a logical approach to the problem of hypertrophy versus strain It is possible that the determination of the ventricular gradient in these cases might clarify the findings Thus, if there is no gradient the T wave changes must be secondary and it would not be necessary to assume additional myocardial changes If, on the other hand, there is a gradient, the T wave changes must be primary and it would be necessary to assume the operation of additional factors such as myocardial ischemia One might then conceive of left ventricular hypertrophy with secondary T wave changes and left ventricular hypertrophy with primary T wave changes In the present state of our knowledge, however, it is questionable that the designation of 'ventricular strain' has advanced us any further toward the truth than a simple statement that the T waves are inverted over one ventricle or another

Right Ventricular Hypertrophy

In right ventricular hypertrophy (Figs 24 to 26) leads obtained over the right ventricle (ordinarily V_1 or V_1 and V_2, sometimes V_{3R}) show an unusually tall R wave which exceeds the S wave in one or more of these same leads There may or may not be depressed RS-T segments or inverted T waves in these leads The duration of the QRS complex in the conventional leads is usually normal but may be prolonged to 0 10 second There is apt to be right axis deviation associated with a vertical position of the heart but this is not invariable The intrinsicoid deflection is generally delayed over the right ventricle, much more often than not the intrinsicoid deflection is actually inscribed later over the right than over the left ventricle As the electrode moves further to the left over the pre-

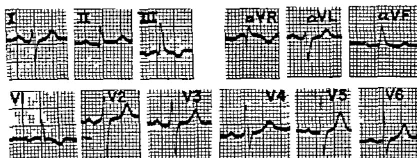


Fig 24. Right ventricular hypertrophy Right axis deviation Lead V_1 shows a minute Q wave a tall R wave a small S wave a minimally depressed RS T segment and an inverted T wave In Lead V_2 there is recorded a large S wave exceeding the R wave in magnitude In Lead V_3 the R and S waves are about equal As the electrode now moves toward Lead V_5 the R wave increases and the S wave decreases It is obvious that Lead V_1 is to the right of the transitional zone Using the criteria enumerated in the text the position of the heart is gen erally indeterminate in right ventricular hypertrophy The patient was a 20 year old girl in whom a murmur was heard during infancy At 10 she had an attack of scarlet fever Examination during a heart survey when these tracings were taken showed a systolic thrill to the left of the sternum a harsh grade III systolic mur mur in the first and second left interspaces and a grade II high pitched systolic murmur at the cardiac apex

cordium the R wave may remain of approximately uniform height or may decrease In making the decision whether right ventricular hypertrophy is present it is important to be certain that the electrode is to the right of the transitional zone If, as rarely happens, there is strong counter-clockwise rotation of the heart on its longitudinal axis (as viewed from the apex) *left* ventricular potentials may be recorded at positions V_1 and V_2 In such cases R waves which are taller than those usually recorded at these points may be recorded at V_1 and V_2 A smooth unbroken pro gression in the height of the R wave from V_1 to V_4 can be detected and at the extreme right position the R wave still exceeds the S wave in mag-nitude In such cases it is important to take additional leads to the right of V_1 to record true right ventricular potentials The change in position

from the left ventricle to a hypertrophied right ventricle may be signaled in an electrode moving from the left to the right precordium by an increase

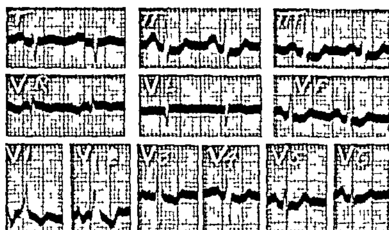


Fig 25 Right ventricular hypertrophy Mitral stenosis Right axis deviation The upper set shows the three standard leads with right axis deviation The middle set shows the three (unaugmented) unipolar limb leads The lowest set shows the six unipolar chest leads The heart is in a semivertical position because Lead V_F resembles Leads V_5 and V_6 and the QRS complex of Lead V_L is small The R waves are tall and the T waves inverted in Leads V_1 and V_2 and decreased from Lead V_2 to V_5 The intrinsicoid deflection is later over the right than over the left ventricle The patient was a thin woman 36 years of age with definite signs of mitral stenosis

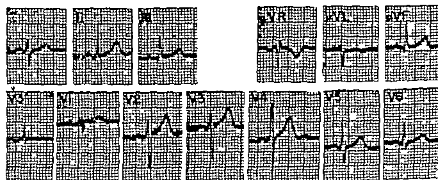


Fig 26 Right ventricular hypertrophy Right ventricular potentials not recorded in usual six precordial leads The diagnosis of right ventricular hypertrophy is not justified on the basis of changes in Leads V_{1-6} but the RR complex in Lead V_1 suggests that the latter is in relation to the transitional zone and that right ventricular potentials have not yet been recorded This prompted the recording of an additional lead further to the right at the midclavicular line (Lead V_{3R} or V_3) this lead shows a tall R wave and an inconspicuous S wave This is decisive evidence for right ventricular hypertrophy

in the height of the R wave after it has decreased or by a changing relationship between the R and S complexes in the leads concerned as the electrode moves toward the right the ratio of the R to the S wave first

decreases, after it crosses the septum onto a hypertrophied right ventricle the R wave again becomes taller than the S wave (Fig 26) A considerable number of patients with right ventricular hypertrophy show a small initial downward deflection which we arbitrarily call a Q wave This change, in association with an R wave which decreases as the electrode moves from the right to the left precordium, could easily mislead the electrocardiographer into making the diagnosis of anterior myocardial infarct (Fig 27) If this lead showing the initial downward deflection is recorded simultaneously with other leads it can be shown that this deflection does not correspond to the very beginning of ventricular activation It probably is actually an S wave preceded by an isoelectric R wave which is not recorded in that particular lead The force responsible for this "Q wave" is probably developed in the apical pole of the right ven-

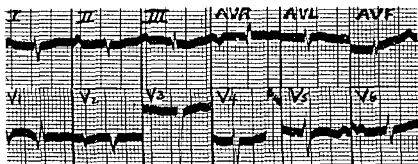


Fig 27 Right ventricular hypertrophy simulating old anteroseptal myocardial infarction ("chronic cor pulmonale") The patient was a 33 year old man with lifelong bronchial asthma who developed right sided congestive heart failure The tracings show low voltage in the conventional leads and a broad Q wave and relatively prominent R wave in Lead V_1 First degree heart block is present The R wave is absent in Lead V_2 and embryonic in Lead V_3 The Q_{V_1} and Q_{V_2} might mislead the electrocardiographer into the diagnosis of anteroseptal infarction but in right ventricular hypertrophy a Q wave may be present over the right ventricle and the R wave may decrease as the transitional zone is approached Postmortem examination showed a thick right ventricle (0 7 cm) pulmonary emphysema peripheral congestion but no myocardial infarct

tricle whose endocardial aspect is faced by the electrode It is important, then, to avoid the diagnosis of anterior myocardial infarction in association with right ventricular hypertrophy on the basis of an initial downward deflection

In our experience the diagnosis of right ventricular hypertrophy made on the above criteria has been invariably substantiated at autopsy In fact, right ventricular hypertrophy is generally apt to be detected by electrocardiography before roentgen ray evidence for right ventricular hypertrophy is present There are a few cases of right ventricular hypertrophy, as verified at postmortem examination, in which the above described electrocardiographic findings were not present during life Some of these show right bundle branch block It has been suggested that in right bundle branch block, the presence of a very tall R over the right

ventricle might be a clue to the additional presence of right ventricular hypertrophy However, tracings of this sort have been induced by catheterization of the right ventricle in a subject with a normal heart, and in another patient with the same type of electrocardiogram the right ventricle was of normal thickness at autopsy This finding of a prominent R′ in right bundle branch block, then, is not conclusive evidence for right ventricular hypertrophy Finally, the empirical observation should be mentioned that *persistent* incomplete right bundle branch block may be considered presumptive evidence of right ventricular hypertrophy The significance of this observation has not yet been clarified

Combined Ventricular Hypertrophy

In most cases of combined ventricular hypertrophy the electrocardiogram shows only the features characteristic of left ventricular hypertrophy

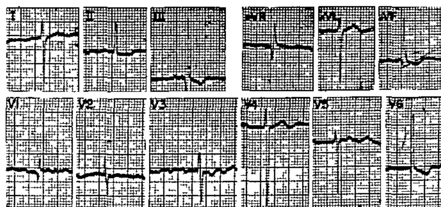


Fig 28 Combined ventricular hypertrophy The patient was a 50 year old woman with rheumatic heart disease mitral stenosis aortic insufficiency auricular fibrillation and congestive heart failure Lead V_1 showed a Q_{V_1} followed by an R wave which was larger than the Q wave An S wave appeared in Lead V_2 and as the electrode was moved to Lead V_5 increased progressively in relation to the R wave In Lead V_6 a tall R wave and an inverted T wave indicate left ventricular hypertrophy The transitional zone was located between Leads V_5 and V_6 On this basis the diagnosis of combined ventricular hypertrophy was made on repeated occasions At postmortem examination there was hypertrophy of both ventricles especially the left Combined ventricular hypertrophy generally cannot be diagnosed electrocardiographically the tracings usually showing left ventricular hypertrophy alone In this case Lead V_1 may actually have recorded left ventricular potentials

In rare instances, however, it is possible to detect electrocardiographic evidence of enlargement of both the right and left ventricles in the same patient (Fig 28) In these cases the tall R wave decreases as the transitional zone is approached, then reappears or increases after the transitional zone has been crossed In many such cases it may be extremely difficult to decide whether one is tapping the potentials of only one ventricle or both The remaining cases of proven combined ventricular hypertrophy are apt to show nonspecific abnormal electrocardiograms, right bundle branch block or evidences of such complicating diseases as myocardial

infarction It seems, then, that combined ventricular hypertrophy is rarely detectable in the electrocardiogram but that the accuracy of this diagnosis is improved if one records with certainty both right and left ventricular potentials

The Clinical Implications of the Electrocardiogram in Ventricular Hypertrophy

At birth the right ventricle is larger than the left and only after several months is the reversed relationship established On the other hand, during the normal aging process the left ventricle often becomes slightly enlarged even when from a practical point of view, it is to be regarded as normal In old age, furthermore as a result of the increased tortuosity of the aorta, the base of the heart is apt to be pushed down and the apex up so that the heart comes to lie in a more horizontal position A heart must not be diagnosed as abnormal simply on the basis of axis deviation to the right or left There are occasions, however, when the electrocardiographic finding of enlargement of one ventricle or the other aids considerably in the differential diagnosis I recall seeing a war veteran who was supposed to have aortic valvular disease because there were a systolic and diastolic murmur and a systolic thrill at the base of the heart The electrocardio gram showed right ventricular hypertrophy This led to an entirely different interpretation, because if hypertrophy develops in aortic valvular disease, it is expected to involve the left rather than the right ventricle More careful examination, including x ray of the heart showed that the condition was some form of congenital heart disease which is often associated with right ventricular hypertrophy Similarly, an elderly woman had congestive heart failure, hypertension and auricular fibrillation The diagnosis was nonvalvular heart disease because there was no diastolic murmur to be heard The electrocardiograms showed well-marked right ventricular hypertrophy This threw some doubt on the diagnosis because left ventricular hypertrophy should have been found with a senile heart and hypertension Mitral stenosis was suspected despite the absence of a diastolic murmur for this could account for the right ventricular enlargement An x ray showed a prominent left auricle and later, on postmortem examination a fish mouth mitral stenosis was found

Axis deviation or ventricular hypertrophy does not always develop according to expectations Disease of the aortic valve and hypertension should produce their effects on the left ventricle by increasing its work Likewise mitral stenosis, congenital disease of the pulmonary valve or pure emphysema should increase the burden on the right ventricle by increasing the pressure in the pulmonary system Although these predicted results generally occur, there are some exceptions that are difficult to explain Until the role of the position of the heart in producing axis deviation was clearly appreciated axis deviation and ventricular hypertrophy were often confused This probably explains many of the discrepancies of the past Even when this possible source of error is eliminated there are still some cases in which the increased load is borne by the left

ventricle and in which prolonged passive congestion in the lungs with increased pulmonary pressure results in hypertrophy of the right ventricle The whole heart in this way may be involved in the burden that one might have thought would be limited to one chamber Such data can, at times, be useful in directing attention to conditions that might otherwise be overlooked It is particularly helpful in appraising valvular conditions, especially when more than one valve is involved Let us assume that a patient has an obvious aortic valvular lesion and shows combined ventricular hypertrophy by electrocardiographic examination This would lead one to suspect the additional diagnosis of mitral stenosis Evidence of right ventricular hypertrophy in the precordial leads is much more likely to develop in congenital heart disease than in mitral stenosis Its frequent absence in mitral stenosis may be due to the relatively slight degree of right ventricular hypertrophy or to overbalance by simultaneous left ventricular hypertrophy

BUNDLE BRANCH BLOCK

An impulse may start in the normal sino auricular node, travel across the auricles, continue down the auriculoventricular node and bundle of His and yet be blocked in the right or left main branch of the bundle If



Fig 29 Left bundle branch block The upper set shows the three standard leads Note that the QRS complex measures 0 15 second and there is no S_1 The lower set shows the six precordial (bipolar) leads from the chest and left leg CF_1 to CF_6 Note that the sharp downward stroke or intrinsicoid deflection (see arrow) comes early over the right ventricle (CF_1 CF_2 and CF_3) and very late over the left ventricle (CF_4 CF_5 and CF_6) The latter QRS complexes often show coarse splitting of the waves as in CF_4 These curves denote delay or block in the left ventricle The patient was a man 53 years of age who had had a coronary thrombosis two years before

the block occurs in the left branch, the impulse descends the right branch normally and reaches the left ventricle in roundabout fashion, probably through the interventricular septum Generally conduction across the

septum consumes about 0 04 second The reverse process occurs if the right branch is blocked The ventricles continue to contract regularly so that there is no arrhythmia in the ordinary sense There is a slight delay, however, in the activation of one ventricle with respect to the other Thus in right bundle block the left ventricle is activated slightly before the right, a reversal of the normal relationship In left bundle branch block, on the other hand, the delay in the electrical activation of the left ventricle is even longer than that occurring in the normally activated heart The ventricular complexes will necessarily be abnormal because of the circuitous route taken by impulse In fact, they will resemble a series of ventricular prema-



Fig 30 Left bundle branch block with auricular fibrillation Note the prominent broad and coarsely notched QRS complexes with oppositely directed T waves These changes are presumptive evidence of left bundle branch block but require confirmation by the detection of late intrinsicoid deflections over the left ventricle In this case the rhythm is irregular because of coexisting auricular fibrillation The patient had hypertensive heart disease and chronic nephritis

ture beats arising from the unblocked ventricle, or the curves seen in ventricular tachycardia The QRS complex is characteristically broadened, coarsely notched, frequently of considerable amplitude and the RS-T segment is apt to be depressed and the T wave to continue in the opposite direction to the main initial deflection In pure experimental bundle branch block and in many clinical instances of bundle branch block measurement of the ventricular gradient shows that these RS-T and T wave changes are secondary' and thus represent changes in the repolarization process contingent upon changes in the depolarization process and are not to be construed as evidence of an altered physiologic state of the myocardium This

point is more fully discussed above in the section on ventricular hypertrophy

When the broadest QRS deflection is upward in Lead I and downward in Lead III the curves represent *classical left bundle branch block* (Figs 29, 30 and 31), and when it is downward in Lead I and upward in Lead III it is *classical right bundle branch block* (Figs 32 and 33) Lead I is generally more helpful than the other two standard leads in determining whether the block is in the right or left branch If there is a prominent S wave in Lead

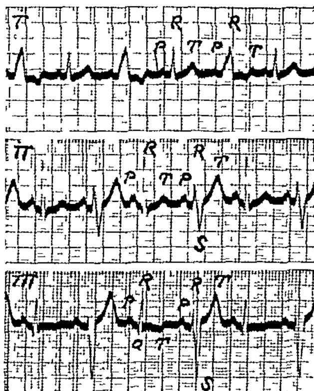


Fig 31 Partial (2 1) left bundle branch block Note that every other ventricular complex has the form similar to Figure 30 The other beats are normal This indicates that every second beat fails to be conducted down the left branch of the bundle of His This patient has hypertensive heart disease Since in contradistinction to the situation in auriculoventricular block the conduction tissue distal to the block is usually stimulated by way of the septum with each impulse this is a very rare phenomenon

I, the block is probably on the right side, if there is a prominent R wave and no S wave in Lead I, it is probably on the left However, in rare instances these relationships will be disturbed because of a peculiarity in the electrical position of the heart This cause of error can be circumvented by examination of the chest leads If the QRS complex, measured in the conventional leads, lasts 0 12 second or longer, and late intrinsicoid deflections are inscribed over the left ventricle, then left bundle branch block is present When we say "late intrinsicoid deflections" we mean that this deflection



Fig 32 Right bundle branch block The upper set shows three standard leads Note that S_1 is broad and QRS = 0 13 second The lower set shows the six bipolar precordial leads from the chest and left leg CF_{1-6} Note that the last sharp downward stroke of the R wave or intrinsicoid deflection (see arrow) comes late in the QRS cycle in CF_1 and CF_2 (over the right ventricle) and comes early in CF_5 and CF_6 (over the left ventricle) This denotes that the block or delay is in the right ventricle The patient was a woman 48 years of age with rheumatic aortic stenosis

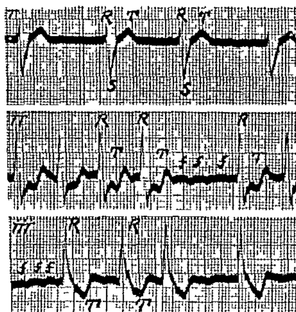


Fig 33 Right bundle branch block with auricular fibrillation Note that the general characteristics of the ventricular complexes (QRS T) are the same as those in Figures 29 30 and 31 but that the main deflections are opposite going down ward in Lead I and upward in Lead III These curves are presumptive evidence of block in the right branch of the bundle of His but should be substantiated by precordial leads showing late intrinsicoid deflections over the right ventricle In this case the rhythm is irregular due to coexisting auricular fibrillation (f f f) At the time these tracings were recorded the patient was 72 years of age had had such curves for at least eight years and showed evidence of well-compensated aortic and mitral stenosis and hypertension He had formerly had frequent attacks of Adams Stokes syncope

is the last part of the QRS complex to be written. In such cases the QRS complex over the left ventricle tends to have a flat or turret top. If these criteria are used, many instances of intraventricular block which do not have the classical appearance in the conventional leads will be classified as bundle branch block, and what looks like left bundle branch block in the conventional leads might, on analysis with precordial leads, actually be revealed as right bundle branch block. Thus in Figure 34 the QRS complex has a duration of 0.14 second, the QRS complex is upright in Lead I and there is no S wave in Lead I, yet the precordial leads clearly show the late intrinsicoid deflection in Leads V_1 and V_2, establishing the site of block in the right bundle.

Unless the situation is complicated by coincident septal infarction a Q wave does not appear over the blocked ventricle. Consideration of the mode of activation of the septum in bundle branch block will make it clear

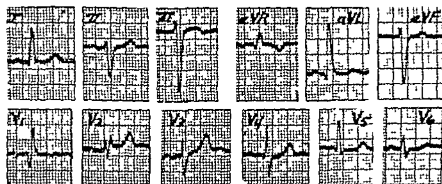


Fig. 34. Right bundle branch block resembling left bundle branch block in the standard leads. The duration of the QRS complex is 0.14 second. Note that there is a small Q wave, a prominent R wave and no S wave in Lead I. T_1 is not inverted so that the curve would not have been considered a "classical" example of left bundle branch block. The precordial leads, showing late intrinsicoid deflections in Leads V_1 and V_2 with R and R' deflections, show that the bundle branch block is on the right rather than, as originally suspected, on the left side.

why this is true. In left bundle branch block the impulse which should travel down the left bundle branch is blocked, therefore the left side of the septum does not get its usual head start in being activated. Instead, the right side of the septum is first activated and the impulse travels from the right to the left side of the septum. Since the impulse moves toward rather than away from the left ventricular cavity, the cavity potential must consist of an initial upward rather than downward deflection. Accordingly those parts of the body which face the left side of the septum no longer record an initial downward deflection or Q wave. This change is illustrated in Figure 35, obtained from a patient with acute posterior myocardial infarction. A well marked Q wave was recorded over the left ventricle when normal intraventricular conduction was present. However, when the process involved the left bundle branch, prolonging the QRS complex to 0.15 second, the Q wave disappeared at those locations where it had pre-

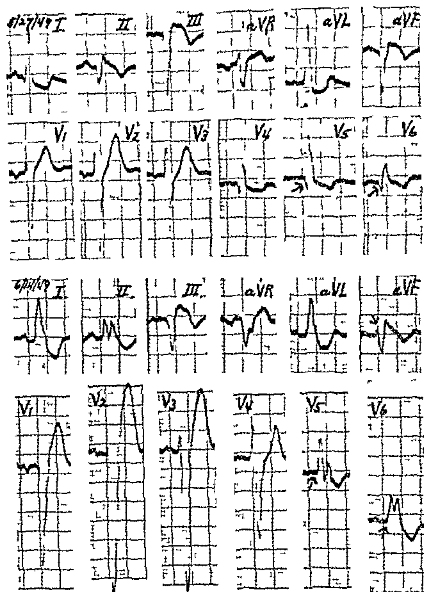


Fig 35 Disappearance of Q wave with development of left bundle branch block The initial set of tracings taken early in the course of an acute clinical episode of chest pain shows the characteristic changes of acute posterior possibly posterolateral myocardial infarct Intraventricular block (QRS = 0 11 *second*) *is* present Note the presence of Q waves indicated by arrows over the left ventricle and in Lead aV_F The second set of tracings recorded eighteen days later shows the development of left bundle branch block The QRS complex now measures 0 15 second Note the late intrinsicoid deflections in Leads V_5 and V_6 Note more particularly the loss of the Q waves in Leads V_5 and V_6 *and also* in Lead aV_F Septal activation now proceeds from the right to the left side of the septum accordingly the left ventricular cavity potential now starts with an upward deflection The decisive evidence of posterior infarction present in the original tracings is therefore lacking in the second set Autopsy showed an acute posterolateral infarct

viously been present Actually about 5 per cent of cases of left bundle branch do show Q waves over the left ventricle In these cases it is necessary to assume infarction of the interventricular septum The infarct, being electrically inactive, permits the potential of the unblocked cavity (in this case of the right ventricle), which is initially negative, to be transmitted across to the blocked ventricle, so that a Q wave may be recorded over the blocked ventricle For similar reasons a Q wave should not be recorded over the right ventricle in right bundle branch block unless septal infarction is present

At times marked left ventricular hypertrophy may produce waves that resemble those of left bundle branch block, but the former will rarely display a QRS interval as long as 0 12 second, will not show such a tardiness of the intrinsicoid deflection in Leads V5 and V6, and may show Q waves in these positions In considering the time of occurrence of the intrinsicoid deflection, due regard must be had for the thickness of the ventricles There is some delay because of the hypertrophy itself, but never so much as results from bundle branch block Although a certain number of patients with ventricular hypertrophy sooner or later develop bundle branch block, practical and theoretical considerations compel us to regard this as a new, emergent and independent development which is not an integral feature in the course of ventricular hypertrophy Ventricular hypertrophy and bundle branch block, then, are two unitary concepts Coincident ventricular hypertrophy and bundle branch block are considered below

Generally the corroborative evidence for bundle branch block will be found with the usual six precordial leads, but it is necessary to be certain that one has recorded true left or right ventricular potentials, preferably at a point where the final moment of activation of the blocked ventricle is recorded, that is, at a point where an S wave is not inscribed In order to accomplish this it is occasionally necessary to record additional leads to the right or left of the usual six precordial points Another note of caution with regard to the diagnosis of bundle branch block is appropriate Even if all the criteria given above are fulfilled it is still impossible to be certain of the existence of bundle branch block unless these ventricular complexes are actually conducted from the auricles With complete auriculoventricular block an idioventricular rhythm arising in the opposite bundle branch may produce ventricular complexes identical with those produced in block of the bundle branch on one side In such cases it is generally wiser to make the noncommittal diagnosis of complete heart block with abnormal ventricular complexes Unless similar ventricular complexes have been demonstrated previously, or are demonstrated subsequently when all or some of these same ventricular beats have clearly been conducted from the auricles, a definitive diagnosis of concomitant complete heart block and bundle branch block is not warranted Since it is generally considered that the irregular ventricular beats are actually transmitted from auricles to ventricles in auricular fibrillation, it seems justifiable to diagnose coexistent auricular fibrillation and bundle branch block (Figs 30 and 33) In occasional cases of this sort, if the ventricular rate is rapid it may be difficult or impossible

to differentiate between ventricular tachycardia on the one hand and bundle branch block associated with auricular fibrillation or flutter on the other (Fig 99)

We have seen that in the normally activated heart septal depolarization proceeds initially from its left to its right side The impulse produces an upward deflection over the right ventricle Subsequent activation of the septum from the right to the left side would produce a downward deflection over the right ventricle but this is antagonized by concomitant and continued activation from the left to the right side of the septum In left bundle branch block where this antagonizing force from the left to the right is lost, the activation from the right to the left is unopposed This eliminates the tendency of the R wave to increase as the electrode moves over the right ventri le from position V_1 to the transitional zone As a pure consequence of this abnormal direction of septal activation in left bundle branch block, the R waves are small or absent over the right ventricle Thus in left bundle branch block the inference of anteroseptal myocardial infarction cannot be made from the presence of small R waves or from the presence of Q waves over the right ventricle Neither can anterior myocardial infarction be excluded under these conditions Accordingly with left bundle branch block one must, from the electrocardiographic standpoint, remain in doubt as to the coexistence of anterior myocardial infarction

Incomplete Bundle Branch Block

There are numerous instances in which there is a delay but no block in one branch or the other, resulting in electrocardiograms resembling those just described except that the duration of the QRS complex is less than

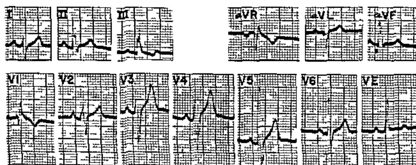


Fig 36 Incomplete right bundle branch block The patient was a 21 year old Navy veteran with a record of a dozen Pacific engagements in whom a heart murmur was discovered on attempting to reenlist Catheterization studies showed the existence of an interauricular septal defect Note that the duration of the QRS complex is 0 11 second that an RR complex is present in Lead V_1 and a notched R wave is present in Lead V_E with late intrinsicoid deflections in both of these leads

0 12 second generally 0 11 or 0 10 second (Figs 36, 37) Such curves have been recorded in the experimental animal during the subsidence of bundle branch block If all of the criteria except the duration of the initial

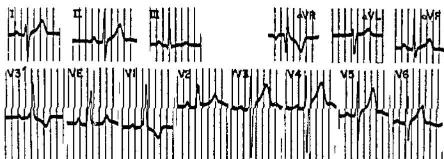


Fig 37 Incomplete right bundle branch block with right ventricular hypertrophy The conventional leads show right axis deviation The zone transitional between right and left ventricular potentials is at Lead V_2 The initial R at Lead V_2 represents left ventricular activation the secondary R (R′) represents right ventricular activation The intrinsicoid deflection is later over the right than over the left ventricle The presence of an initial R rather than Q wave over the right ventricle is more in accord with incomplete right bundle branch block than right ventricular hypertrophy but the tallness of R′ suggests the additional existence of right ventricular hypertrophy The patient was a 13 year old schoolboy with a grade IV systolic murmur and thrill in the third and fourth left intercostal spaces X ray examination showed right ventricular hypertrophy and a dilated pulmonary artery Catheterization studies established the existence of pulmonic stenosis

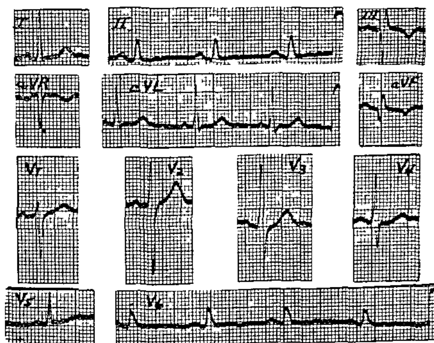


Fig 38 Incomplete left bundle branch block The patient a 54 year old man with a typical attack of acute myocardial infarction developed the characteristic changes of an acute posterior lesion associated with a QRS interval of 0 11 second and a late intrinsicoid deflection in Lead V_6 Note that there is no Q wave in Leads V_{4-6} or aV_L The patient recovered

ventricular complex are fulfilled the diagnosis of incomplete bundle branch block is warranted The exact meaning of this finding is still under investigation Incomplete right bundle branch block may occur as a transient finding in acute myocardial infarction and in acute cor pulmonale, and as a persistent finding in right ventricular hypertrophy An appreciable number of individuals with hearts normal in all other respects show incomplete right bundle branch block In making the diagnosis it is important to be certain that the electrode is not higher than the fourth interspace else the potentials of the pulmonic conus may be recorded Since the pulmonic conus is one of the last parts of the heart to be activated an electrode in relation with this structure may show a late intrinsicoid deflection and thus falsely raise the question of incomplete right bundle branch block

Nor is the significance of incomplete left bundle branch block settled In all individuals whose electrocardiograms show the characteristic appearance of left ventricular hypertrophy but which lack a Q wave over the left ventricle, the possibility of this condition exists (Fig 38) As with complete left bundle branch block the abnormal activation of the septum from the right to the left in incomplete left bundle branch block may explain the failure of the R wave to increase over the right ventricle as the electrode moves to the left which finding may lead to the false inference of anteroseptal infarction

Bundle branch block is often associated with other types of heart block such as auriculoventricular or sino auricular block (Figs 107 119) In Figure 31 is represented a rare instance of 2 1 partial left bundle branch block At first glance one might interpret this as coupled rhythm due to premature ventricular systoles, but on careful inspection it will be found that auricular and ventricular beats come exactly on time

Clinical Implications of Bundle Branch Block

Bundle branch block is fairly common in general practice Recent experience with unipolar chest leads has shown, contrary to previous opinion that block on the right side is much more frequent than block on the left side Whatever is actually the more frequent the use of the term common type of intraventricular block should be discarded as inexact and equivocal

Inasmuch as bundle branch block occurs for the most part in patients with regular rhythm it is difficult to recognize this condition without electrocardiograms Attempts to detect this condition at the bedside or even to verify anatomic interruption of one bundle branch or the other with serial microscopic sections have often been futile Yet neither failure refutes the incontrovertible electrical evidence of delayed activation of one ventricle or the other Bundle branch block is a purely electrocardiographic diagnosis However it is often possible to suspect its presence Whenever a diastolic gallop rhythm is heard one should think of the possibility of bundle branch block Moreover, bundle branch block is often accompanied by a bifurcated or reduplicated apex impulse Both of these findings need to be looked for carefully and deliberately for when they are detected, bundle branch block is apt to be present Finally, in such cases pulsus

alternans is also very common. In fact, the frequent association of bundle branch block, gallop rhythm and pulsus alternans should lead one to suspect the presence of one if the other two are found.

Bundle branch block occurs most frequently in conjunction with hypertensive heart disease, with disease of the coronary arteries, in cases of aortic stenosis and much less so in association with mitral stenosis. It almost always denotes some disease of the myocardium, although there are rare instances in which no other clinical evidence appears that the heart is diseased. The prognosis for patients with left bundle branch block is poor. The average length of life after it is first noted is not more than a year or two but there are exceptional patients who carry on fairly satisfactorily for many years. The situation is quite different in right bundle branch block, for here many patients continue in good health for a great many years. In fact, there are instances of this type in which there seems to be very little other evidence of organic disease and the prognosis may be extremely favorable. There is no specific treatment for persons with this disturbance as it does not, by itself, produce any handicap. It merely reflects the condition in the heart muscle and therapy is directed at the general state of the circulation.

Intraventricular Block

Complete and incomplete bundle branch block are, strictly speaking, special forms of intraventricular block. We have been accustomed, how-

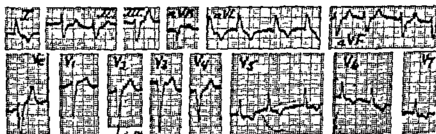


Fig. 39. Intraventricular block. The QRS complex in the standard leads measures 0.14 second. Although there is a superficial resemblance to left bundle branch block, it will be noted that in Leads I, aV_L and V_{5-7} the QRS complex is not flat or turret topped and does not have a true intrinsicoid deflection in the sense of a final rapid downward deflection. The descending limb of the R wave begins relatively early in the QRS complex and shows a smooth, slow oblique slope. Such curves should be designated intraventricular block. Note also the absence of clearly demonstrable P waves. The patient was a 58 year old spinster admitted for uremia and acidosis. The electrocardiogram suggested potassium intoxication. No postmortem examination was performed.

ever, to make the diagnosis of intraventricular block by exclusion when bundle branch block cannot be established. Thus if, with a QRS duration of 0.11 second or longer, it is impossible to demonstrate late intrinsicoid deflections over one ventricle or the other, one must, by exclusion, make the noncommittal diagnosis of intraventricular block or defective intraventricular conduction (Figs. 39 and 40). Some of these latter hearts will,

on further analysis, show localized areas of delayed activation If the transitional zone can be eliminated as the cause of notching of the QRS complex in one or more precordial leads and if flanking leads show earlier activation, one may be dealing with "focal" block Occasionally this local area of delayed activation may be restricted to esophageal leads recorded at ventricular levels This finding probably signifies localized patchy fibrosis, probably involving the subendocardial Purkinje network The term "arborization block" might be used as the equivalent of focal block but this should not be confused with the "arborization block" of the older literature

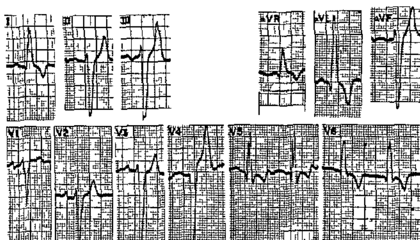


Fig 40 Left ventricular and septal hypertrophy with pronounced intraventricular block The patient was a 19 year old man with coarctation of the aorta The tracings show a QRS interval of 0 16 second a duration unusual for uncomplicated left ventricular hypertrophy Late intrinsicoid deflections over the left ventricle and flat topped R waves in Lead V_6 suggest left bundle branch block However deep notching of V_5 of the type seen here is not usually observed in left bundle branch block and the presence of Q waves in Leads V_5 and V_6 is incompatible with pure left bundle branch block At postmortem examination the heart was tremendously enlarged (930 gm) the left ventricle measuring 2 2 cm in thickness There was a congenital bicuspid aortic valve The septum though strikingly hypertrophied was the site neither of infarction nor of other disease There was some scarring in the lateral wall of the left ventricle most marked subendocardially The septal hypertrophy may be the principal cause of the prolonged QRS interval and the subendocardial scarring of the Q waves over the left ventricle

Analysis of many of the tracings showing bizarre low voltage prolonged QRS complexes, formerly considered as evidencing "arborization block" would, in the light of the criteria given above, demonstrate may of them to be examples of bundle branch block

The clinical implications of defective intraventricular conduction are similar to those of bundle branch block It should be borne in mind that generally a grave condition of the ventricular musculature is indicated and the prognosis should be guarded accordingly Occasionally, such curves will be found when there is little else to make one suspect heart disease

and in that way they become very helpful diagnostically In fact, the greatest value of electrocardiography is in the detection of significant abnormalities in the ventricular complexes when the rest of the examination reveals no essential abnormality

DISTURBANCES OF THE PACEMAKER (SINO AURICULAR NODE)

Normal Tachycardia

It appears that the sino-auricular node has to go through some chemical process in building up material which finally explodes and sends out an impulse This process normally repeats itself in a fairly orderly fashion at a rate of about 70 per minute There are numerous common conditions in



Fig 41 Normal sinus tachycardia Upper tracing is from a boy, aged 9, who had a postscarlatina tachycardia rate 143 Middle curves are from a boy aged 18 with neurocirculatory asthenia rate 146 Note all complexes are normal in sequence but merely come at a rapid rate The T_2 and T_3 in neurocirculatory asthenia are frequently flat or inverted Lower set is from a boy, aged 9 with acute rheumatic heart disease, rate 166

which the process takes place more rapidly, i e, exercise, emotion, fever or hyperthyroidism Under these circumstances the impulse starts in the normal focus and travels across the heart normally but the rate at which it is repeated is increased This is called *normal tachycardia* or *sinus tachycardia* (Fig 41) All the complexes have an essentially normal configuration and most of the acceleration takes place at the expense of the diastole of the heart (the T-P interval) Such a condition need not indicate disease of the heart It was doubtful whether any organic heart disease was present in the patient illustrated by the upper curves of Figure 41 and there certainly was no heart disease in the patient from whom the middle tracings were obtained At times it is very important to distinguish a normal tachy-

cardia from one due to an ectopic rhythm and it may be necessary to produce vagal stimulation as is illustrated in Figures 66, 74 and 112 When a rapid regular rhythm is due to an abnormal mechanism, vagal stimulation either produces no effect, stops the tachycardia or causes temporary abrupt alterations in the rate while in normal tachycardia the effect is slight or gradual

Normal Bradycardia

The vagus and sympathetic control have much to do with the regulation of the rate of impulse formation at the pacemaker Under certain conditions, either as a result of increased vagal or diminished accelerator tone, the rate of the heart is unusually slow and sluggish, 45 or less (Fig 42) This occurs in some normal healthy individuals, particularly tall young athletes with undernutrition, as a result of jaundice, during sleep and after certain infections The entire mechanism of the beat is normal and, therefore the electrocardiogram will be normal in every respect except that the diastolic pauses will be great The condition is called *normal bradycardia* At times the rate can be below 40 and even 35 with a normal mechanism At these low levels it muct be clearly distinguished from heart block, for with the former the heart is apt to be normal and with the latter the heart



Fig 42 Normal bradycardia Rate about 42 from a man aged 68 who had no symptoms or signs of heart disease Note that the complexes are normal the rate is slow and there is a sinus arrhythmia

is almost always diseased This can readily be done without special apparatus for on exercise the rate will gradually rise to a higher level and then return to the original slow rate if the bradycardia is a normal one, whereas when heart block is present the rate will either change slightly or not at all or sudden interruptions in the length of the heart cycle will be detected

Sinus Arrhythmia

There is an irregularity of the heart called *sinus arrhythmia* in which gradual acceleration and retardation take place (Figs 43 and 44) It is sometimes called respiratory arrhythmia because it often is phasic with respiration The rate speeds up with inspiration and then slows with expiration At times it is independent of breathing The impulse originates in the normal pacemaker and traverses the heart normally, the gradual changes taking place mainly in the length of diastole Because it is so common in childhood it is also called juvenile arrhythmia When it is marked or when the changes are abrupt it may be confused with more serious irregularities such as auricular fibrillation or heart block It occurs in many healthy

people, in some older individuals with myocardial disease and after full digitalization It is apt to disappear entirely if the heart rate is increased artificially by exercise or by diminishing the vagal tone with atropine The

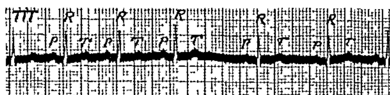


Fig 43 Sinus arrhythmia A woman aged 38 who had malnutrition but no heart disease Note gradual increase and decrease of the length of the heart cycles The changes take place almost entirely in the length of the diastolic pause (T P interval)

important point is that it must be regarded as an essentially normal phenomenon and does not indicate organic disease When the heart rate has been rapid and regular for some cause, such as hyperthyroidism or rheu-

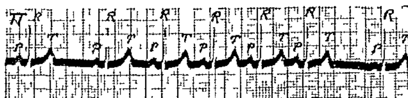


Fig 44 Sinus arrhythmia From a boy 6 years of age with a normal heart Note more abrupt changes in the length of the heart cycles

matic carditis, the appearance of sinus arrhythmia is apt to indicate that the condition is progressing favorably and the normal vagal control is returning In fact, it is rare to find this arrhythmia while active thyrotoxicosis or rheumatic carditis exists

Sinus Pauses

The pacemaker of the heart may become inhibited as a result of certain reflex influences and thereby fail to produce impulses for varying lengths of time Under such circumstances the whole heart fails to contract and if the pause is sufficiently long, giddiness or actual syncope occurs, presenting clinical features that are similar to those seen in Adams-Stokes disease This condition is called *sinus pauses* (Fig 45) The electrocardiographic complexes are normal in form but there appear long diastolic pauses of varying lengths This might be regarded as an exaggerated form of sinus arrhythmia The mechanism of this disturbance depends on a reflex stimulation of the vagus such as a vagovagal or carotid sinus reflex Figure 45 is a tracing of a patient who had spells of unconsciousness precipitated by the act of swallowing or gagging and accompanied by a peculiar sensation in the throat It was found that each time a tongue depressor was applied to the tongue to examine the pharynx an attack occurred during which the heart stopped for several seconds

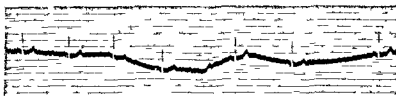


Fig 45 Sinus pauses Note marked irregular slowing of the heart neither auricle nor ventricle contracts This resulted from vagus stimulation following gagging (probably a vagovagal reflex) The irregularity of the base line is an artefact and due to movement of the patient (Author s article in Oxford Loose Leaf Medicine vol II)

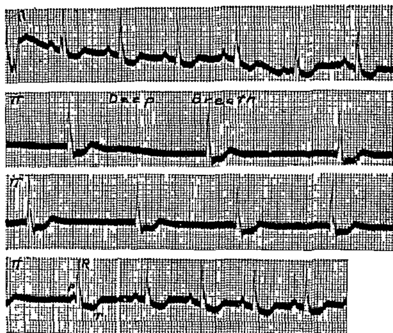


Fig 46 Sinus pauses This is a continuous tracing from a man with aortic stenosis and angina pectoris Note that on taking a deep breath the heart slowed markedly the P waves disappeared there was an idioventricular rhythm and a gradual return to normal

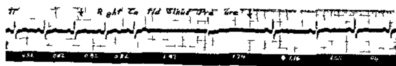


Fig 47*A* Sinus pauses From a man aged 60 who had syncopal attacks but no heart disease The right carotid sinus was very sensitive Note marked slowing of the heart from the carotid sinus reflex

Similar electrocardiograms and syncopal attacks can occur in some individuals with aortic stenosis or in those who have a sensitive carotid sinus Figure 46 shows the effect of a deep breath in a man with aortic stenosis

The whole heart slowed and the patient grew somewhat faint Figure 47A shows how readily the heart can be slowed by light pressure over the carotid sinus in some individuals This man was quite well except that he had fre-

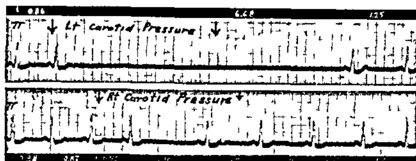


Fig 47*B* Marked carotid sinus sensitivity Male 60 years old with hyper tension aortic stenosis and angina pectoris Note the prolonged asystole of over *six seconds following left carotid pressure The effect is less marked on the right*

quent spells of unconsciousness which came without warning He was entirely cured by taking $\frac{3}{8}$ grain (0 025 gm) of ephedrine sulfate two or three times a day Another instance of marked carotid sensitivity is shown in Figure 47*B*

Auricular Standstill

On rare occasions electrocardiograms are seen in which the auricular complexes entirely disappear for brief or considerable periods of time This

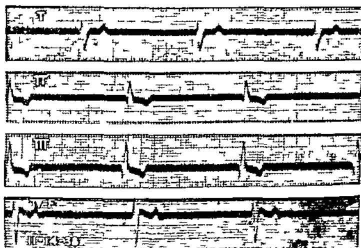


Fig 48 Auricular standstill Male 70 years old who had taken excessive digitalis and developed unconscious spells Note complete absence of P waves and ventricular rate of 33 Patient made complete recovery with reappearance of normal P waves

condition is called auricular standstill or inhibition of the auricles (Figs 48 and 49) The ventricular beat is maintained by the idioventricular pace-

maker Such curves are occasionally observed during quinidine or digitalis administration and indicate a toxic effect of the drug Syncopal or Adams-Stokes attacks may occur in patients manifesting this disturbance and it is

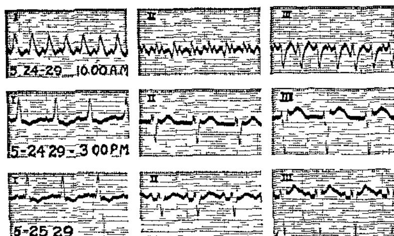


Fig 49 Auricular standstill due to quinidine Upper tracing shows paroxysmal ventricular tachycardia with a ventricular rate of 169 Middle set shows auricular standstill in Leads II and III Lower set shows return of auricular activity The patient had received a single dose of 1 gm of quinidine sulfate at 11 30 a m on May 24 1929

imperative that the drug causing the auricular standstill be omitted Auricular standstill is also apparently a regular feature of the more advanced stages of potassium intoxication

ECTOPIC RHYTHMS

In the preceding paragraphs we discussed disturbances in the pacemaker of the heart The second major abnormality in the mechanism of the heart beat is the formation of ectopic rhythms Impulses can arise in almost any part of the heart auricles, junctional tissue or ventricles When such ectopic beats occur they interfere with the normal sequence of events and produce peculiar and characteristic electrocardiographic changes The fundamental principle underlying these alterations is that if an impulse travels an abnormal course through a certain portion of the heart, the electrocardiographic representation of that impulse will be abnormal

Premature Auricular Beats

Impulses may arise in any portion of the auricular musculature Ordinarily the tendency for beats to arise in abnormal parts of the heart is held in abeyance because the pace set by the sino auricular node is faster and prevents other foci from functioning Under abnormal conditions ectopic foci are enabled to initiate impulses and when such isolated beats occur they are called *premature* or *ectopic beats* or *extrasystoles* On listening over the precordium one hears a regular rhythm (lub-dub, lub dub) and then

suddenly a quick beat followed by a pause, after which the regular sequence is restored This premature beat may come only at very rare intervals or as frequently as every second or third cycle Although the beat can be heard over the precordium it may produce such a small pulse wave that it is only barely felt at the wrist or it may be entirely imperceptible This is so because the heart contracts early in diastole when the volume of blood in the ventricles *is quite small*

From an electrocardiographic point of view we must visualize the wave of excitation as arising in some point in the auricle more or less distant from the *sino auricular node* The course through the auricle that this impulse must take will be abnormal It will travel possibly from left to right rather than from right to left or upward rather than downward The P wave which represents auricular activity will have to be abnormal in form in

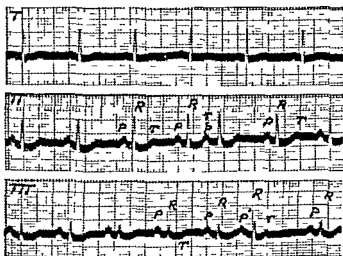


Fig 50 Premature auricular beats Note that the irregularity is due to an occasional premature beat (P) which becomes superimposed on the previous T wave The ventricular complexes following the premature beats are of normal form (Leads II and III) From a man who had a benign irregularity of the heart.

one or more leads How different from the normal P wave it will be will depend on how far away the ectopic focus is from the normal pacemaker It may, therefore, remain upward or become inverted (Figs 50, 51, 52) When this impulse reaches the sino auricular node it destroys whatever impulse forming material had been built up there and the node starts over again in the production of a normal beat The premature beat is also traveling downward through the auriculoventricular junctional tissue to reach the ventricles The pathway of this beat through the ventricles therefore should be normal and the QRS-T complex will be of normal form Generally this is so (Figs 50 and 51) At times the ventricular complex following a premature auricular beat, however, is *abnormal (Fig 51)* and the P-R interval may be delayed In fact, on rare occasions the beat may be blocked and then it is called *blocked premature auricular beat* This occurs because

the beat has come so quickly that the tissue has not recovered completely and there may be a slight delay in one part or another of the conduction apparatus distorting the spread of the wave of excitation The premature and abnormal P wave may need to be sought for carefully It often is hidden on the previous T wave and is to be detected by a slight alteration in the height or configuration of this T wave (Fig 50) At other times because the ectopic focus is so near the normal pacemaker the P waves differ only very slightly from the normal ones (Fig 92, lower curves) Occasionally a premature auricular beat produces no electrical disturbance in one of the leads but the finding of a P wave of an abnormal form in one of the other leads will reveal its abnormal origin

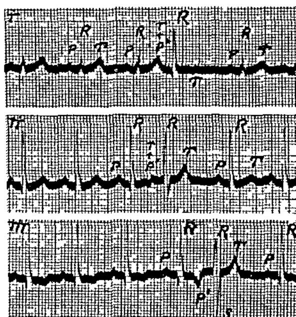


Fig 51 Premature auricular beats From a young man with no heart disease Note that the premature P waves fall on the preceding T waves are inverted in Leads II and III and are followed by ventricular complexes of abnormal form

Premature auricular beats can readily be distinguished from those of ventricular origin Even when the former are followed by abnormal ventricular complexes, the QRS waves are not so broad as in the latter condition The detection of the preceding abnormal P wave identifies the beat as auricular Finally, the pause after an auricular extrasystole, although longer than the normal cycle, is generally not completely compensatory, i e , the length of the two cycles including the extrasystole is less than two normal heart cycles, whereas with ventricular extrasystoles it is equal to two normal beats Occasionally, however, even premature auricular beats are followed by completely compensatory pauses The detection of a compensatory pause is not, therefore, a reliable method of differentiating ventricular from auricular premature beats

Premature auricular beats are fairly common and do not by themselves indicate heart disease They occur in individuals otherwise well as a purely functional or neurogenic disturbance and need produce no symptoms or disability When such is the case, the irregularity should not be treated, nor should the patient be restricted in his activities When these beats pro-

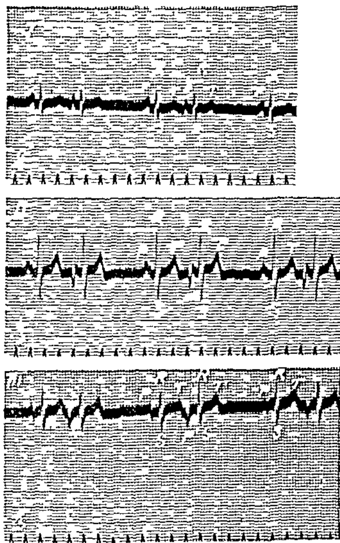


Fig 52 Premature auricular beats Note that every second beat is a premature P wave which is upright in Lead I but inverted in Leads II and III This is a form of coupled rhythm (Author s article in Oxford Loose Leaf Medicine vol II)

duce a good deal of palpitation and are annoying, quinidine or digitalis may be helpful Occasionally potassium salts (potassium phosphate or chloride, 2 gm three times a day by mouth) may prove beneficial Unlike ventricular extrasystoles premature auricular beats do not result from excessive digitalis They do occur in patients with organic heart disease, but the diagnosis of structural disease will then have to rest on other evi-

dence There is some association between this type of extrasystole and auricular disturbances of higher grade, e g , tachycardia, flutter and fibrillation Patients who show the latter irregularities often are found to have premature auricular beats at other times On following the progress of a case of mitral stenosis this form of extrasystole often occurs for some years preceding the development of auricular fibrillation, and so the detection of the former may lead one to suspect that the latter irregularity will not be long delayed Likewise, if a patient has paroxysms of some form of rapid heart action, the finding of an occasional auricular extrasystole between attacks helps to throw light on the nature of the paroxysms that have occurred in the past

Paroxysmal Auricular Tachycardia

As one continues the consideration of ectopic rhythms in the auricle a disturbance of somewhat higher degree is paroxysmal auricular tachycardia (Figs 53 to 64) Here instead of an occasional impulse arising in some abnormal part of the auricular wall this focus sends out a series of regular impulses The pace is then set by this ectopic focus and it may be main-

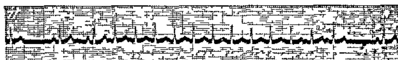


Fig 53 Paroxysmal auricular tachycardia Note the complete cycle of a brief attack of tachycardia lasting five seconds P is different in form from the normal P wave but the ventricular complexes are unchanged The onset and offset are instantaneous the rhythm is perfectly regular rate is 158 The patient was a girl of 21 without heart disease who complained of palpitation

tained for variable lengths of time, persisting for hours or days and rarely for weeks A very new concept concerning the nature of paroxysmal auricular tachycardia is developing at present It is now thought by some observers that a circus movement which has its pathway through the auriculoventricular or sino auricular node is responsible for this abnormality Inasmuch as the origin of the impulse is abnormal the course it takes through the auricles is abnormal and in so far as an auricular complex can be identified it will differ from the normal P wave The course of this wave through the ventricles, however is normal and therefore the QRST waves will be the same as when the heart is beating normally Figure 53 represents a complete brief paroxysm of this type lasting only four to five seconds It shows that the onset and offset are abrupt and the auricular waves change their form when the paroxysm starts, but that the ventricular complexes remain the same It is characteristic of this condition that the rhythm is perfectly regular much more so than in a normally beating heart It is also peculiar that in most cases the P waves cannot be easily identified for they probably are fused with the preceding ventricular complex The result is that one often sees only what appears to be an R and T wave coming in rapid succession

Clinically this condition is important as it is common, can cause a good deal of discomfort and concern and on rare occasions can have serious con-

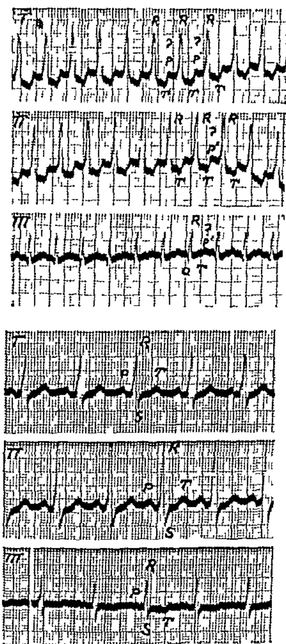


Fig 54 Paroxysmal auricular tachycardia Upper three leads show a perfectly regular rate of 172 Auricular complexes (P) probably buried in the previous T waves Lower curves on the same patient taken after attack was over show a normal slow rhythm rate of 83

sequences It is also important from a practical point of view because it can readily be recognized at the bedside and effectively treated It occurs

for the most part in an individual who has a structurally normal heart Occasionally it is a complication of organic heart disease and then may produce alarming symptoms Ordinarily attacks occur without any detectable cause The patient is apt to blame some particular happening that took place just before the attack such as a motor ride or eating a certain kind of food Indigestion or gas is often suspected as being the cause, and yet treatment directed at the gastrointestinal tract or the diet will be of no avail A careful history will frequently bring to light the fact that a certain specific event may act as a trigger in setting off a paroxysm A sudden turn of the head or bending over to tie one s shoes, an unexpected emotional upset or a dream all have been known to precipitate such spells On the other hand many attacks occur without any obvious cause

The attack itself is instantaneous in onset and offset (Fig 53) In most cases the patient is aware of this and will describe it as coming suddenly and stopping with a ' thump ' At times, although the changes are sudden, the sensations will be described as occurring quickly but not abruptly The patient will complain of palpitation or fluttering of the heart become uneasy nervous and apprehensive and want to lie down Occasionally there is pain over the heart and there may be typical anginal distress even with radiation to the arms Sometimes nausea and vomiting occur and with it attacks often end This sort of experience convinces the sufferer that it is all due to indigestion or to some food that was eaten After the attack has lasted a variable length of time, it suddenly stops and the patient quickly recovers Many will feel perfectly well shortly afterward while a few will be left weak enough to remain in bed a day or so How distressing the symptoms will be and how severe the evidence of circulatory failure will depend on three factors—the duration of the attack the rate of the heart during the attack and the state of the heart before the attack occurred A normal heart can withstand a rate of over 200 continuously for several days, while if mitral stenosis is present, evidence of congestive heart failure may develop in hours

The *clinical diagnosis* of this condition is simple in most cases It is important to obtain a history or elicit evidence of an abrupt change in rate The heart rate during an attack of paroxysmal auricular tachycardia ranges from about 150 to 250 per minute, although slower rates may occasionally be found *The rhythm is perfectly regular By this is meant that* contiguous heart cycles will not vary more than 0 01 second in length (Fig 57) This is very important diagnostically for the ear is able to detect very minor alterations in rhythm Not only is the heart rapid and regular but its rate is very fixed for long intervals of time and cannot be altered by simple procedures like breathing or exercise which affect the rate of the normal heart Over the course of hours the rate may change but not during short intervals of time The bedside application of this peculiarity is to count the rate carefully for sixty seconds by auscultation over the precordium This should be repeated in several minutes, preferably under different circumstances, after slight exercise or with the patient sitting up rather than lying down If there is a significant difference in the two rates

one can be fairly sure it is not paroxysmal auricular tachycardia With the latter the first count might be 169 and the second 168 If the second count were 174, it would point to a normal sinus tachycardia (Fig 41) The difference should be no greater than two beats if the count is made carefully Even this difference is to be accounted for by the difficulty in timing the first and last beats as one follows the second hand of the watch in its complete circuit One often hears the statement that the rate, though regular, was too rapid to be counted Although this may be so when the rhythm is irregular, I believe it means careless observation or lack of interest in accurate counting when it refers to a rapid regular rate I had the opportunity of testing this in a case that showed a heart rate of 250 Three independent observers counted rates of 248, 249 and 250 When the rate is over 200 the actual counting process may be facilitated by tapping on the table with a pencil synchronously with the heart beat and then counting the taps Emphasis is placed on this because determining the constancy of the rate can be carried out by any physician and helps to distinguish a normal from an abnormal tachycardia

The various methods that are used to stop an attack also serve as diagnostic procedures, for there is no other type of rapid heart action that can be made to return to a normal rate by such simple means and so quickly Not only does a normal tachycardia reach its rapid rate gradually, starting from about 70 or 80 and then increasing to 100, 120 and finally 160 or 170, but when it returns to normal it does so gradually If vagus stimulation is produced by pressure over the carotid sinus or the eyeball during paroxysmal tachycardia, the rate will remain entirely unaltered or it will abruptly fall to normal, i e, the rate may change from 190 to 80 in one or two beats (Figs 55, 56, 57, 74) With normal tachycardia, there is apt to be temporary slowing with gradual return to the previous rapid level (Figs 74, 112) A similar effect is generally obtained if the tachycardia is due to auricular flutter (Fig 66) and when ventricular tachycardia is the cause of the acceleration vagus stimulation produces no effect whatever It is evident, therefore, that the diagnosis can generally be made at the bedside and only rarely will electrocardiograms be necessary

The *treatment* of the condition can be divided into two problems The first and easier task is to stop the attack The second and more difficult is to prevent its recurrence Most attacks will end if left alone and no harm will result Occasionally severe congestive heart failure or anginal pain may develop and rarely may prove to be fatal For thirty years I have followed one extraordinary patient who had yearly severe attacks in 1911, 1912, and 1913 During one of these he developed aphasia, which cleared in several months, during another he had a hemiplegia which disappeared in a few months leaving only a very slight spasticity, and during the third, dry gangrene requiring amputation of the left arm at the shoulder developed The tachycardia in these spells lasted from five to eleven days incessantly When I saw the patient during his fourth attack the heart rate was 250 The blood pressure was about 94 mm systolic and 88 mm diastolic He developed a fever of 101 and an appreciable leukocytosis He also complained

of a constant distressing constricting pain in the chest There were a few rales at the bases of the lungs Many of these features resemble those of an acute coronary thrombosis, a condition with which any severe form of paroxysmal rapid heart action can be confused In fact, the heart was found to have dilated 2 5 centimeters as compared to its size on x ray examination

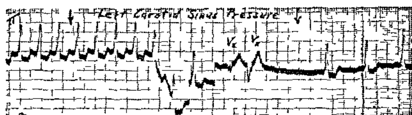


Fig 55 Paroxysmal auricular tachycardia (same case as Fig 54) Note the abrupt cessation of the attack by pressure over the left carotid sinus The irregularity of the baseline is due to movements of the patient Vx indicates ventricular premature beats which are common during the transitions Patient had hypertension and angina pectoris

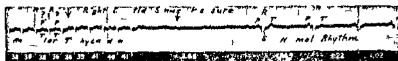


Fig 56 Paroxysmal auricular tachycardia Cessation of attack by right carotid sinus pressure Note long pause followed by resumption of normal rhythm Rate of attack 158 P waves during attack not easily identified Patient had no heart disease

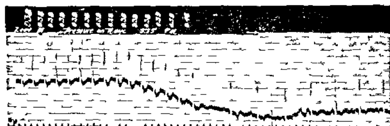


Fig 57 Paroxysmal auricular or nodal tachycardia Cessation of attack by right ocular pressure (oculocardiac reflex) Note sudden transition from regular rhythm with rate of 158 to rate of 80 Numbers above tracing measure the inter ventricular intervals in hundredths of a second Note absolute regularity with slight slowing just before the transition The treatment of auricular and nodal tachycardia is the same Patient had rheumatic aortic insufficiency (Author s article in Oxford Loose Leaf Medicine vol II)

after the attack was over Such dilatation is unusual in paroxysmal tachycardia, for in most cases there is no dilatation or very little It is quite evident that proper treatment was very urgent in the case just cited At first it was found that carotid sinus pressure was ineffective but vigorous pressure over the eyeball ended the attack immediately Subsequent attacks were readily controlled by pressure over the carotid sinus and the patient

was taught how to perform this procedure For some unknown reason the attacks gradually became quite rare The patient remained in excellent health for over twenty years but lately has had typical mild anginal pain on effort which is not associated with attacks of tachycardia

Further illustrations of the urgency of proper treatment are the rare instances in which attacks occur in patients on the operating table during anesthesia Breathing may cease entirely and the patient may become pulseless Even fatalities may occur I have seen several cases in which the condition seemed critical but was readily controlled by simple methods

The simplest means of stopping an attack is to have the patient hold a deep inspiration as long as he can He might be advised to make a quick expiratory effort with the glottis closed after a deep breath is taken Many attacks can be ended quite readily in this way The patient should be told that if this fails, he might try to induce vomiting by placing his finger down the pharynx The act of gagging, retching or vomiting often ends attacks This may be brought about by the hypodermic injection of apomorphine

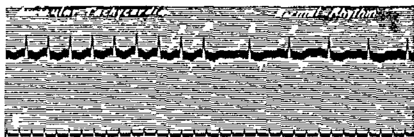


Fig 58 Paroxysmal auricular tachycardia Cessation of attack following 0 3 gm of quinidine sulfate given intravenously Abrupt transition occurred about thirty seconds after injection from rate of 181 to 97 The patient had carcinomatosis but no heart disease (Author s article in Oxford Loose Leaf Medicine vol II)

More effective still are 1 to 4 teaspoonfuls of syrup of ipecac Some have learned that lying down or lowering the head may be effective Often when the attack continues, morphine is given and during the subsequent hour or two the heart returns to normal When this occurs it is difficult to say that the medication causes the attack to end The most valuable treatment is carotid sinus pressure This is performed by placing two fingers over the carotid artery just below the jaw, where a slight bulge is felt as the common carotid artery divides, and pressing and massaging the artery against the spine for several seconds First one side and then the other may be tried but not both at the same time This stimulates the vagus nerve reflexly (carotid sinus reflex), although it used to be thought that the vagus nerve which lies just behind the artery in the carotid sheath was stimulated directly In the majority of cases, when this is properly done, the attack will end instantly If this fails, ocular pressure may be tried (Fig 57) One eye at a time should be firmly pressed backward To be effective the pressure will have to be painful, but this method may succeed where others have failed It is particularly applicable if the patient is already under an anes-

thetic for then the pain will not be felt In this procedure the reflex goes up the fifth cranial nerve and down the vagus (oculocardiac reflex) There have been rare instances in which carotid sinus pressure has resulted in hemiplegia I have seen one such case in an elderly man Very likely when marked cerebral sclerosis is present the fall in blood pressure and cessation of the circulation may result in local cerebral thrombosis In fact, in extremely rare cases, fatalities have occurred because after a pause in the heart the normal beat was not resumed

Finally, there are drugs that may stop attacks of tachycardia Recently acetyl β methylcholine or mecholyl has been found to be very effective

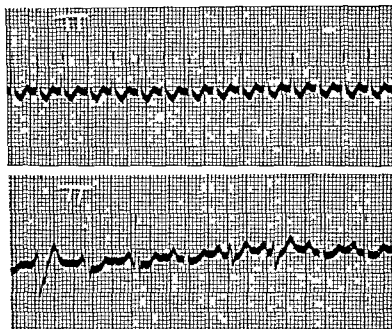


Fig 59 Paroxysmal auricular tachycardia arrested by mecholyl Upper tracing shows regular rate of about 225 Lower set taken one minute after 25 mg of mecholyl was injected subcutaneously shows slow rate with occasional extrasystoles The patient was a woman 36 years old with no *organic heart disease* Note electrical alternation of the R waves during tachycardia

(Fig 59) The average adult dose is about 20 mg, given subcutaneously This produces a rather powerful stimulating effect on the vagus apparatus It often aborts an attack in a few minutes Because of occasional untoward reactions atropine should always be available for immediate use in doses of 1 to 2 mg given hypodermically Asthmatics or strongly allergic individuals should not be given mecholyl Another drug that I have employed in former years when all other methods failed and the condition of the patient seemed critical is quinidine sulfate administered intravenously (Fig 58) The dose that was used was 0 3 gm (5 grains) There is some danger of instant fatality from the intravenous use of quinidine so it cannot be

recommended unless the circumstances warrant the risk Until recently quinidine preparations have not been available for parenteral use The fol-

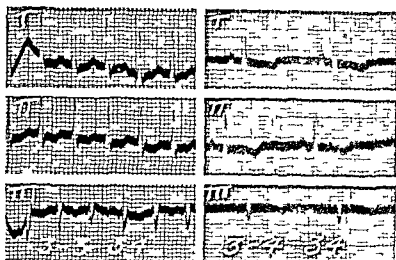


Fig 60 Paroxysmal auricular tachycardia stopped by digitalis Tracings on the left show regular rate of 196 Set on the right taken twenty hours later, 1 gm of digitalis having been given orally shows normal rate of 69 The patient was a man 62 years old with bronchopneumonia and a fever of 103 F who recovered satisfactorily

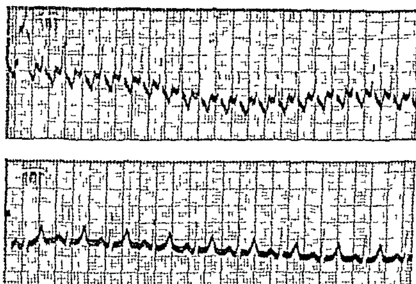


Fig 61 Paroxysmal auricular tachycardia stopped by magnesium sulfate Upper tracings show regular rate of 257 Lower set was taken two minutes after the intravenous injection of 3 gm of magnesium sulfate and shows a normal rhythm The patient was a man 68 years old with probable spinal cord tumor but no heart disease

lowing procedure, however, could be employed A mixture is made of 0 3 to 0 5 gm of quinidine sulfate in 20 to 30 cc of water To this a few drops

of concentrated hydrochloric acid must be added to keep it in solution After boiling the solution may be slowly injected intravenously It has also been suggested that the required amount of quinidine may be dissolved in 200 to 300 cc of saline This slower method of injection may be safer Ampules containing 0 6 gm of quinidine hydrochloride have now been prepared and are suitable for intramuscular or intravenous use Frequently large doses of digitalis (0 5 to 1 0 gm given parenterally or orally) may gradually arrest an attack (Fig 60) Recently magnesium sulfate in doses of about 2 0 gm given intravenously has also proved successful (Fig 61) There is a slight risk with this method as there is with all intravenous medication for tachycardia Finally, prostigmine in doses of 0 5 to 1 0 mg subcutaneously has been effective in 10 to 15 minutes, especially if followed by carotid sinus stimulation In general it will be found that the paroxysm can be controlled by one method or another

The prevention of attacks presents a more difficult problem If they recur infrequently, once a year or so it hardly seems wise to institute a course

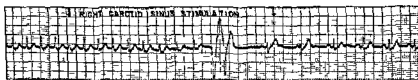


Fig 62 Paroxysmal auricular tachycardia Recording of Lead II showing supraventricular tachycardia with rate of 192 At first sight the small sharp *inverted P waves deforming the RS T segments immediately after the QRS* complexes seem to be retrograde from the auriculoventricular node to the auricles Attention to the last beat in the paroxysm terminated with carotid sinus stimulation shows no such inverted P wave following the QRS complex It is clear then that the inverted P waves are not conducted back from the ventricles but rather are conducted to the ventricles after a prolonged P R interval A pair of ectopic ventricular beats interrupt the paroxysm are followed by a pause then the resumption of normal sinus rhythm at a rate of 108

of drug therapy because not knowing when the attack is due it will be necessary to keep the patient on the medication all those intervening months with the hope of inhibiting this rare spell It is better to do nothing and then to stop the attack when it occurs When attacks come frequently the problem is different for they may be very annoying and it is possible to ascertain whether or not drug therapy is effective Occasionally the constant administration of 0 2 to 0 3 gm (3 to 5 grains) of quinidine sulfate will prevent attacks Even more effective is constant complete digitalization This must be carried out just as it is done in congestive heart failure One or the other of these methods frequently obviates or at least diminishes the frequency or severity of the attacks

Although in most cases, heart block is not associated with paroxysmal auricular tachycardia there is a small group in which auriculoventricular block of some degree does exist (Fig 63) This type differs from the more usual one in that it is less responsive to the measures that ordinarily stop attacks, it may persist for long periods of time even months or years, and

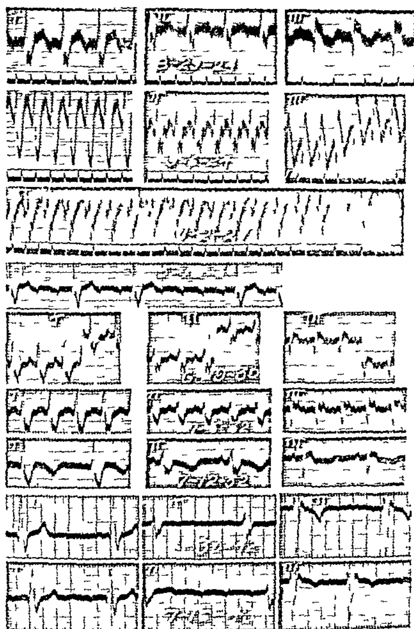


Fig 63 Paroxysmal auricular tachycardia with block (unusual type) Series of tracings taken over a course of nineteen years Some show a 1 1 rhythm with a ventricular rate of about 240 (second set) Others show a 2 1 rhythm (first set) with ventricular rate about 120, the fourth and the last sets show varying block with auricles still rapid regular at about 200 and ventricles slow at about 50 interspersed are sets with normal slow rhythm (seventh and eighth) Right bundle branch block is present throughout The patient is now 72 years old and has been ambulatory without congestive failure all this time

it presents some features that resemble auricular flutter In fact, recent studies of this and other types of paroxysmal auricular tachycardia lend support to the idea that some of the cases are due to a circus movement in

which the pathway runs through the auriculoventricular node It will be found that quinidine and digitalis are only occasionally effective in con-

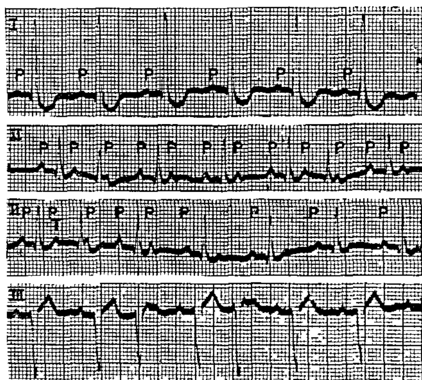


Fig 64 Paroxysmal auricular tachycardia with block Normal sinus rhythm in Lead I with heart rate 89 and P R interval 0 18 second In the second strip (Lead II) paroxysmal auricular tachycardia (auricular rate 176) with block (ventricular rate 107) is recorded Note the isoelectric period between the P waves contrasting with the constantly moving baseline characteristically seen in auricular flutter The third strip a continuation of Lead II shows cessation of the paroxysm of auricular tachycardia Note the irregularity of the ventricles during the paroxysm and the regularity of the ventricles during normal rhythm and the contrast between the sharper taller P waves with tachycardia of the auricles and the lower more rounded P waves with normal rhythm In the fourth strip (Lead III) the abnormal rhythm is resumed The patient was a 63 year old man with calcific aortic stenosis and aortic insufficiency angina pectoris and congestive heart failure who had been treated with digitoxin 0 1 to 0 2 mg daily over a two months period associated with nausea vomiting diarrhea and photophobia After seven days without digitoxin the abnormal rhythm and the other symptoms disappeared

trolling this rarer form of tachycardia, although digitalis may at lea[illegible] the ventricular rate

Auricular Flutter

A still higher degree of auricular disturbance is auricular [illegible] 65 to 73) In this condition the auricular rate generally is be[illegible]

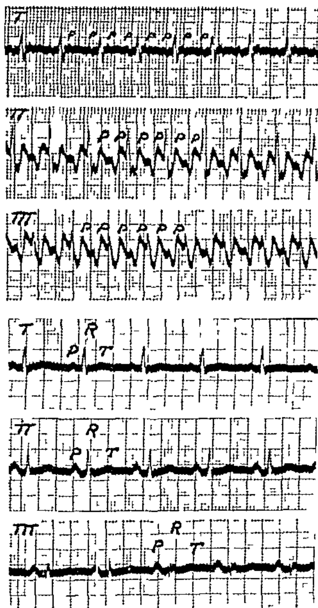


Fig 65 Auricular flutter Upper three leads taken during auricular flutter lower tracings taken four weeks later during normal rhythm During tachycardia the auricular rate is 278 ventricular 139 (2 1 block) Identity and rapidity of auricular beats are difficult to make out until the ventricles are slowed (see Fig 66) Note the small P waves in Lead I, prominent triangular P waves in Leads II and III with a sharp upstroke and a coarsely notched downstroke Normal mechanism was established on quinidine therapy The patient had no heart disease but complained of syncopal attacks for three years

350 and except in very rare instances the ventricles respond to only a fraction of this number, as the junctional tissue cannot conduct impulses so rapidly, i e , there is in a sense a certain degree of auriculoventricular block

In exceptional cases all auricular beats come through to the ventricles and the heart rate is then very rapid (Fig 69) During quinidine therapy the auricular rate may fall to as low as 150 It has been shown that the mechanism of this peculiar abnormality is the development of a circus movement By this is meant than an impulse gets started and encircles the auricles around the venae cavae and on its return to the original focus finds the tissue out of its refractory state and continues around again and again, always meeting muscle bundles ahead of it that are ready to conduct the impulse In auricular flutter this pathway is constant and therefore the waves will be regular and constant in form In auricular fibrillation, to be discussed later because the rate is more rapid, each impulse finds bits of tissue ahead of it that are still refractory following the previous contraction, so that it has to deviate to one side or another constantly seeking fibers that will conduct The result is a sinuous course and more irregular and more inconstant fibrillary waves occur Auricular flutter has therefore been regarded as a pure circus motion while auricular fibrillation has been regarded as an impure one The validity of this concept has long been denied by continental writers Recent cinematographic and electrographic studies on

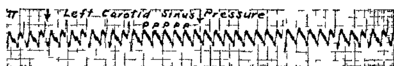


Fig 66 Auricular flutter Effect of left carotid sinus pressure (Same patient as Fig 65) The ventricular rate is temporarily inhibited permitting the undisturbed rapid auricular mechanism to become clear

experimental flutter in this country by Printzmetal have cast further doubt on the traditional teaching

The electrocardiograms of auricular flutter are very peculiar and characteristic In Lead I the auricular waves are represented by small notches (Fig 65) In Leads II and III the waves have a triangular form The up stroke is sharp and smooth and the downstroke more prolonged and notched at its midpoint It seems as if there is a hesitation on the downstroke where the isoelectric line might have occurred It makes one think that each auricular cycle begins at this point and that the entire complex is diphasic rather than beginning either at the top or bottom of these P waves The flutter waves are continuous, as one cycle ends the next begins There is also a similarity between the flutter waves in different patients In untreated patients there will generally be found a 2 1 heart block, every second beat failing to reach the ventricles The auricular rate may be 320 and the ventricular 160 It therefore may be difficult to distinguish the auricular waves or count their rate because they become superimposed on the ventricular complexes With experience one learns to detect flutter from the appearance of the electrocardiograms but if there is any doubt as to the underlying mechanism vagal stimulation can help to identify the condition (Fig 71) In Figure 65 it may be questioned whether there are

one or two auricular complexes between each ventricular beat However, when the ventricles are inhibited for a short period of time by pressure on the carotid sinus (Fig 66) or by digitalis (Figs 67 and 68) it is then possible

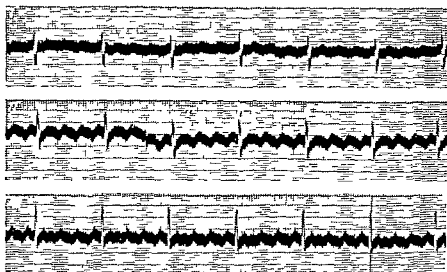


Fig 67 Auricular flutter The P waves are regular, rate 208 the ventricular complexes are regular rate 52 there is a 4 1 block Note triangular form of P waves in Leads II and III The slow ventricular rate resulted from digitalis (Author s article in Oxford Loose Leaf Medicine vol II)

to see the flutter waves undisturbed The true rapid rate easily becomes apparent and is found to be twice the original ventricular rate The continuous activity of the auricles is also readily visualized from such tracings

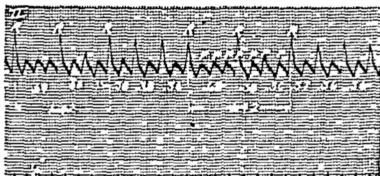


Fig 68 Auricular flutter The ventricular beats (R) come irregularly but because of responses to regularly beating auricles (P) there is equal grouping of beats i e not an absolute irregularity (Author s article in Oxford Loose Leaf Medicine vol II)

The rhythm of the auricles is strikingly regular The ventricular rate and rhythm will depend on the degree of heart block When there is a 2 1 block, as is commonly the case, the ventricles will necessarily be perfectly

regular contiguous cycles not varying more than 0.01 second in length, just as in paroxysmal auricular tachycardia. We now have three conditions

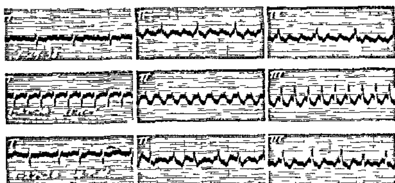


Fig. 69. Auricular flutter. Upper curves show mainly a 3:1 block. Middle set shows a 1:1 rhythm. Both auricular and ventricular rates are 191. Lowest set shows a changing degree of block.

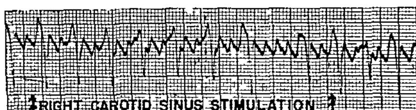


Fig. 70. Auricular flutter with extremely rapid flutter rate. The patient was a 54 year old woman who developed auricular flutter with a flutter rate of 440 and a 3:1 response during a thyroid storm. Note slowing on right carotid sinus stimulation disclosing characteristic sharp ascent and interrupted descent of the flutter waves. Extremely rapid rates in this range are only rarely recorded.

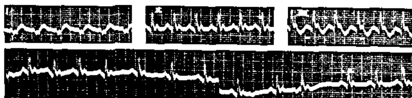


Fig. 71. Value of carotid sinus stimulation in deciphering an abnormal auricular rhythm. The patient was a 68 year old man with an acute myocardial infarction. The electrocardiograms (upper set) showed a ventricular rate of 136. An upward deflection particularly well seen in Lead II was interpreted by one observer as auricular flutter by another as paroxysmal auricular tachycardia with block. Right carotid sinus stimulation (lower tracing) shows that what was thought to be a P wave was actually part of the QRS complex. Precordial leads showed this secondary R wave to be an evidence of right bundle branch block.

that might account for a rapid regular heart rate, i.e., normal tachycardia, auricular tachycardia and auricular flutter. It often is possible to distin-

guish these at the bedside If the regular rate that is counted at the apex is 190 or more, the condition is most likely paroxysmal tachycardia, for a normal tachycardia rarely reaches that high level, the condition is not likely to be auricular flutter because the auricular rate would have to be 380 which is too fast, or 190 which is not only too slow but would necessitate a 1 1 mechanism which occurs but is extremely rare (Fig 69) I have only rarely seen instances of flutter with auricular rates of 380 to 440, twice during a thyroid storm (Fig 70) and once during lobar pneumonia It must be remembered that on auscultation only the ventricular beats are heard, as the auricular are inaudible Furthermore, if vagal stimulation produced by breathing, carotid pressure or ocular pressure terminates the rapid rate completely the condition must be paroxysmal tachycardia for neither normal tachycardia nor flutter ever respond in this fashion If the ventricular rhythm is temporarily disturbed and slowed, the diagnosis of auricular tachycardia is eliminated for in the latter the tachycardia either would be unaffected or would disappear entirely When the ventricular rate is slowed, therefore, the condition may still be either flutter or normal acceleration (Figs 66 and 112) The length of the heart cycles during these interruptions, the method by which the rapid rate is resumed and other factors may then distinguish the one from the other During the long pauses if frequent auricular waves can be seen in the jugular pulse, auricular flutter is present Similarly this will be the diagnosis if the heart rate at any time, even if for very short intervals, is found suddenly to be halved Figure 74 illustrates *the response of various types of rapid heart action to vagus stimulation* produced by carotid sinus pressure From such observations one is enabled to distinguish one type from another by simple bedside examination

When digitalis is given to a patient with auricular flutter the original degree of block increases Instead of every second, only every fourth beat may reach the ventricles (Fig 67) The ventricular rate will now necessarily be perfectly regular and slow If we assume the original auricular rate to have been 300, and the ventricular 150, the auricular rate will be the same but the ventricular 75 When a patient is seen for the first time under such circumstances the underlying mechanism can easily be overlooked for one would not suspect that the rhythm were abnormal with a regular rate of 75 If one thought about the possibility of auricular flutter, one might search for and detect numerous auricular waves in the jugular veins because the auricles are contracting constantly Of greater help than this is to have the patient exercise a bit The heart rate may be found to jump suddenly to exactly twice its former rate for a short time, i e , the original 2 1 block returns, or an irregularity will develop that will lead to the proper diagnosis

A further effect of digitalis is that the ventricular rate may become irregular The degree of block then varies Sometimes every second beat or every third or fourth beat comes through to the ventricles (Fig 68) The irregularity may seem to be gross and resemble the total irregularity of auricular fibrillation On careful examination, however, it will be found that there still remains a dominant rhythm, as the ventricles respond to regularly beating auricles Grouping of beats will occur that are of equal lengths,

inasmuch as they correspond to equal numbers of auricular cycles (Fig 68) This distinguishes it from auricular fibrillation

Unlike paroxysmal tachycardia which generally occurs in otherwise normal hearts, auricular flutter is apt to be associated with organic heart disease, either valvular or myocardial although it can manifest itself as a purely functional disturbance It may occur in the form of paroxysms lasting hours or days and be transient, but more often once established it is permanent unless the patient is treated It can persist for many years It occasionally develops during coronary thrombosis, hyperthyroidism,

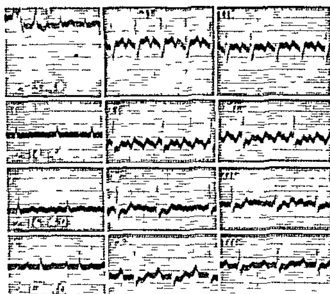


Fig 72 Auricular flutter (response to digitalis) Upper set shows auricular flutter with 2 1 rhythm auricular rate 300 ventricular rate 150 second set shows flutter with 4 1 rhythm auricular rate 300 ventricular 75 third set shows auricular fibrillation with ventricular rate about 75 lowest set shows normal rhythm rate about 95 The patient received 0 4 gm digitalis orally on February 13 1940 and 0 6 gm on February 14 1940 then digitalis was omitted This patient was 63 years old had mitral stenosis and insufficiency with marked dyspnea and palpitation

rheumatic fever, pneumonia, pericarditis or other infections, or as a terminal event in chronic nephritis

The aim in the *treatment* of patients with auricular flutter is to restore the normal rhythm or, if this is impossible, to keep the ventricular rate slow When digitalis is given, in about one third or a half of the cases the following changes will take place First the ventricular rate slows, the flutter continuing Then after several days the mechanism changes to auricular fibrillation If the digitalis is omitted at this point, a normal mechanism may quickly be reestablished (Fig 72) Often the flutter continues on digitalis therapy with a slow ventricular rate or the fibrillation that develops persists It is preferable to have the latter rather than the

former condition Quinidine is also a valuable therapeutic agent in auricular flutter On increasing doses the auricular rate will actually slow, at times to well under 200, while the effect on the ventricles will vary The ventricular rate may be slowed or accelerated With the diminished rate of the auricles a larger proportion of the beats may reach the ventricles

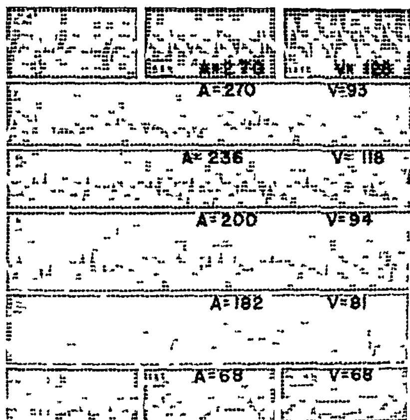


Fig 73 Effect of quinidine on auricular and ventricular rates in auricular flutter The patient was a 38 year old man without evidence of organic heart disease who complained of spells of palpitation for three years occasionally associated with syncope *A* Leads I II and III on admission before quinidine Auricular flutter with 2 1 response Auricular rate 270 *B* Second day On digifolin Auricular flutter with varying (2 1, 4 1) response Auricular rate 270 *C*, Fourth day after increasing doses of quinidine (0 5 to 1 5 gm) for two days Auricular rate 236 Ventricular rate 118 Note that the ventricles have speeded up while the auricle have slowed This is a frequent finding and apt to be alarming but the drug should be continued despite this *D* Fifth day Auricular rate 200 *E*, Seventh day Auricular rate 182 *F* Tenth day Still on quinidine Normal sinus rhythm rate 68 Note slowing of auricular rate from 270 to 182 before reversion to normal rhythm

(Fig 73) Finally the circus motion in the auricles may be broken up and a normal mechanism established At times large doses of quinidine are necessary to accomplish this, even as much as 1 5 or 2 gm (22 to 30 grains) in a single dose (Fig 73) It is advisable to try digitalis first, as it is safer and if it does not accomplish the desired effect, it is better to have the

patient digitalized before giving quinidine Patients with auricular flutter receiving drug treatment require careful observation and should be in a hospital where the condition can be followed with frequent electrocardiographic records

Auricular Fibrillation

The highest degree of auricular disturbance is called auricular fibrillation In this condition the number of auricular impulses is very great 350

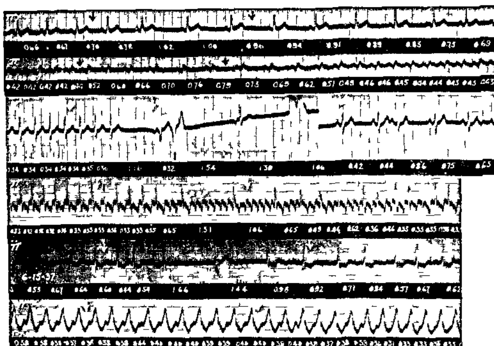


Fig 74 Effect of vagal stimulation Arrows in each tracing indicate duration of carotid sinus pressure Numbers indicate duration in seconds of respective heart beats First set shows normal gradual slowing in *normal rhythm* Second set shows gradual slowing with gradual return in *normal tachycardia* Third set shows instant cessation of an attack of *paroxysmal auricular tachycardia* Fourth set shows temporary slowing of the ventricles with irregular return to the original rapid rate in *auricular flutter* Note that the auricular rate remains unaffected Fifth set shows temporary slowing of the ventricles in *auricular fibrillation* with return of original grossly irregular rhythm Last set shows no effect in *ventricular tachycardia* slight irregularities persisting throughout These effects characterize each type of mechanism

or more The mechanism, as has been described, is due to a circus motion in which the path is impure and irregular The auricles do not actually contract, but rather remain distended in diastole and show fibrillary twitching There is no constant pathologic change in the auricles to explain auricular fibrillation and at present it is regarded as a functional derangement accompanying a variety of states The condition can be easily repro-

duced in the experimental animal by faradizing the auricles and I have had the opportunity of seeing it in the living human heart in a patient with adherent pericardium who was undergoing a pericardial operation The number of auricular impulses is so great that only a portion of them can be conducted through the junctional tissue Therefore, there is always

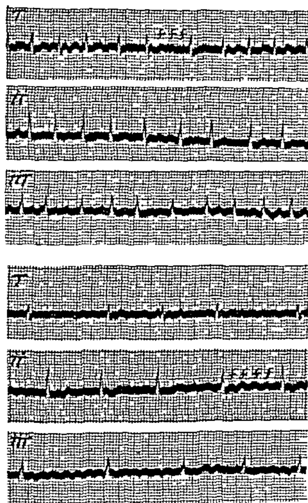


Fig 75 Auricular fibrillation Effect of digitalis The upper three leads were taken July 9 1935 Note the gross irregularity of the ventricular beats (rate 147) the absence of the normal P wave and the presence of the auricular fibrillary waves (f f f) rate 391 The lower three leads taken July 10, 1935 after 1 5 gm of digitalis were given show fibrillation continuing but the ventricular rate is now 60 and still irregular The patient had mitral stenosis and decompensation

some degree of heart block associated with auricular fibrillation The ventricular response is grossly irregular, short cycles and longer cycles occurring *without rhyme or reason The peripheral pulse is necessarily irregular* both in time and in force of contraction In the past it has been called perpetual arrhythmia, absolute or total irregularity, and delirium cordis In

more recent years the true nature of this disorder has been clarified and the term 'auricular fibrillation' given to it

It follows from the preceding description that whatever results from the normal presystolic contraction of the auricles would disappear when fibrillation develops In the electrocardiogram the normal P wave will be absent and instead there will be found irregular rapid fibrillary waves (f f-f) for the most part throughout the cardiac cycle, as the auricular activity is continuous There will also be a totally irregular ventricular response (Figs 75 to 82) Inasmuch as the impulses that succeed in reaching the ventricles travel down the normal auriculoventricular conduction path the ventricular complexes are normal in form These three features of the electrocardiogram, i e, the absence of the P wave, the presence of fibrillary waves and

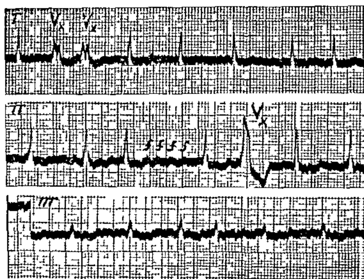


Fig 76 Auricular fibrillation Note the absolute irregularity of the ventricles the rapid fibrillation of the auricles (f f f) and the absence of the normal P waves V indicates ventricular extrasystoles The patient had no heart disease and the rhythm became regular on increasing doses of quinidine

the gross irregularity of ventricular beats make it quite simple to identify auricular fibrillation Sometimes the fibrillary waves are quite prominent, especially in mitral stenosis, and they may resemble those seen in auricular flutter It would seem as if the circus motion in places becomes almost perfectly regular Furthermore, these fibrillary waves may be entirely absent in one or more leads (Fig 77 Lead III) Occasionally the diastolic interval is perfectly smooth in which case the diagnosis of auricular fibrillation will rest on the other criteria or on taking esophageal, precordial or special auricular leads Finally under certain circumstances, although the auricles are fibrillating the ventricles contract regularly (Fig 82) In some cases when digitalis is administered this takes place It is thought that complete heart block results and while the auricles continue fibrillating

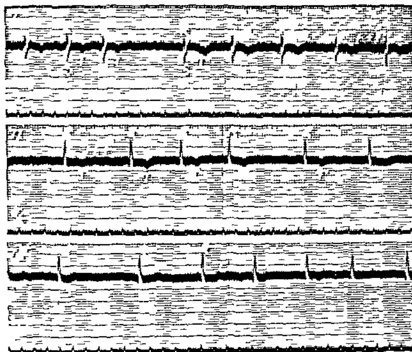


Fig 77 Auricular fibrillation Note the absolute irregularity of the ventricles and the absence of the P waves The fibrillary waves (f f f) are practically invisible in Lead III and very small in Leads I and II The patient had mitral stenosis and decompensation (Author s article in Oxford Loose Leaf Medicine vol II)

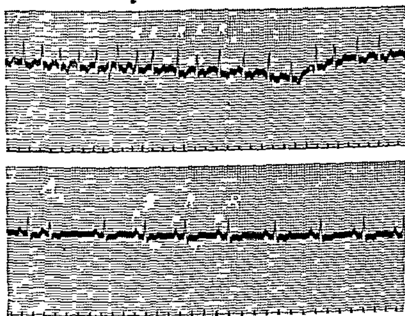


Fig 78 Auricular fibrillation paroxysmal The upper tracing shows typical auricular fibrillation with an irregular ventricular rate of 157 The lower tracing taken a few days later shows normal auricular contractions (P waves) rate 85 There is one premature auricular beat (A_x) (Author s article in Oxford Loose Leaf Medicine vol II)

the pace is set for the ventricular rate at the auriculoventricular node or junctional tissue When this takes place as a result of digitalis, the regular ventricular rate of complete heart block is not the slow one customarily seen in Adams-Stokes disease On the contrary, the rate will be 55 to 70

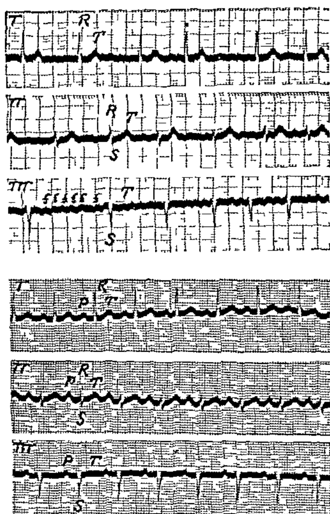


Fig 79 Auricular fibrillation Effect of quinidine Upper three leads taken June 19 1935 show typical auricular fibrillation Lower three leads taken June 28 1935 show a normal rhythm Increasing doses of quinidine sulfate up to 0 5 gm, had been given in the meantime Contrast this with the effect of digitalis Figure 75 This patient had no organic heart disease

or more and on increasing the dose may exceed 100 In fact, if digitalis is continued in large doses a fatal intoxication may result The important inference from this is that when a patient with auricular fibrillation develops a regular rhythm while taking digitalis the change may be due either to a

resumption of the normal rhythm or to the development of this peculiar type of complete heart block On rare occasions true spontaneous complete heart block with a ventricular rate of 30 may be associated with auricular

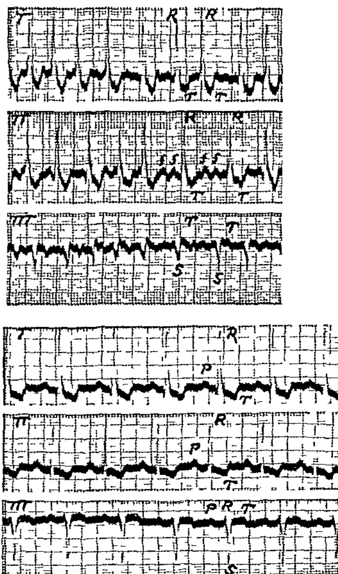


Fig 80 Auricular fibrillation Effect of quinidine Upper three leads are typical of auricular fibrillation lower three leads show a normal rhythm Regularization occurred the morning after three doses of 0 3 gm of quinidine sulfate were given The patient was a man 25 years old with mitral stenosis and aortic insufficiency

fibrillation This subject is further considered below in the discussion of digitalis toxicity

From a clinical point of view auricular fibrillation is the most important of all disturbances in the mechanism of the heart beat It is extremely com-

mon it produces considerable disability if untreated and responds very satisfactorily to treatment It occurs in the transient (Fig 78) as well as in the permanent form When paroxysms occur they generally last several hours or a day or so When the irregularity has lasted for about a week it may be expected to persist indefinitely unless specific measures are taken to restore the normal mechanism The most common conditions with which it is associated are rheumatic valvular disease with mitral stenosis, hypertensive heart disease, coronary artery disease and hyperthyroidism It occasionally develops suddenly during acute infections like pneumonia and rheumatic fever It is common during the early days following an acute coronary thrombosis Gallstones are not infrequently found in some of the elderly individuals who have the transient form of auricular fibrillation, but whether there is any association between the two conditions is not certain On rare occasions auricular fibrillation develops as a result of digitalis therapy Finally, it may be present either as a paroxysmal or as a permanent phenomenon in otherwise healthy individuals This latter group of patients

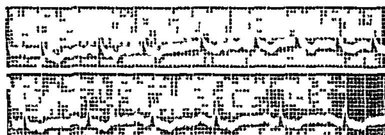


Fig 81 Auricular fibrillation Effect of propylthiouracil Upper curve shows auricular fibrillation in Lead II when patient was thyrotoxic The lower curve was recorded one month later when evidence of thyrotoxicosis had disappeared and normal sinus rhythm had been restored following the daily oral administration of propylthiouracil 200 mg daily

is an important one and curiously most of its members are males Paroxysmal auricular fibrillation is more common than is generally supposed It is particularly characteristic of hyperthyroidism and may be the single clue that leads one to this diagnosis In fact it is wise to suspect hyperthyroidism whenever a transient spell of this irregularity occurs

The bedside recognition of this condition is generally simple A rapid apparently grossly irregular heart with an appreciable pulse deficit (the count at the apex being 10 beats or more greater than at the wrist) is due to auricular fibrillation nine times out of ten The common association between a past history of rheumatic fever or chorea, the presence of mitral stenosis, and auricular fibrillation enables one to predict the presence of one of these three factors if the other two are known to exist If a patient had rheumatic fever and shows signs of mitral stenosis then a grossly irregular heart is almost always due to auricular fibrillation If this irregularity is present and there is a history of rheumatic fever, there probably is mitral stenosis Finally, if the patient has mitral stenosis and auricular fibrillation,

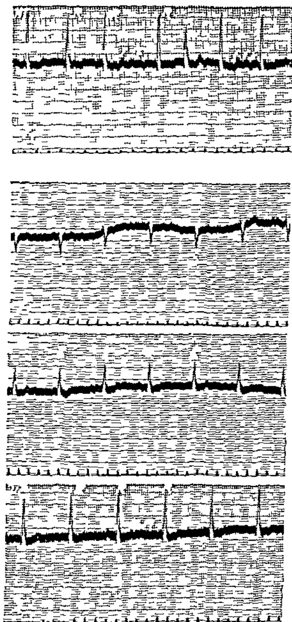


Fig 82 Auricular fibrillation with regular ventricular rhythm The upper curve shows the typical gross irregularity The lower three leads were taken after complete digitalization and show that the fibrillation continues although the ventricles are perfectly regular i e probably complete heart block of the toxic type due to digitalis (Author s article in Oxford Loose Leaf Medicine, vol II)

he has been rheumatic whether a positive history can be obtained or not Exceptions will be found for these generalizations but it is surprising how frequently they will be valid

When auricular fibrillation first develops the ventricular rate is quite

rapid except in rare instances in which some defect in the junctional tissue already is present When digitalis is given it slows the ventricular response Figure 75 shows such an effect the ventricular rate falling from 147 to about 60 in twenty four hours after a large dose of digitalis was administered It must be borne in mind that the fibrillation of the auricles persists Digitalis does not stop the fibrillation, it merely prevents many of these rapid beats from reaching the ventricle through its vagal influence on the conduction tissue Physicians are too inclined to use the expression that the fibrillation is less when what is meant is that ventricles are slower or less tumultuous The fibrillation of the auricles is either present or absent, from a practical point of view there are no degrees of fibrillation When the ventricular rate slows however the pulse deficit diminishes and may entirely disappear At this time the bedside rule of thumb is no longer diagnostic If a physician first sees a patient under these circumstances, the diagnosis is more difficult and must be made on other criteria Irregularities of the heart beat and of the pulse though slight will still be present The condition may resemble other arrhythmias Exercise may accelerate the rate and make the more characteristic features return Rarely will it be necessary to have electrocardiograms before a final decision can be made There is one additional clinical finding that may help to distinguish fibrilla tion from a very irregular rhythm due to numerous extrasystoles In both conditions cycles of short, long or medium duration may occur at various times In irregular rhythm due to extrasystoles it will be found that with extrasystoles all the long pauses are preceded by quick beats because they are compensatory The same auscultatory phenomenon is present with auricular fibrillation However, if one listens long enough and hears a sudden pause not preceded by a quick beat, this points to auricular fibrillation (See the fifth heart cycle in Lead II, Fig 77) In other words, a sudden lengthening of the heart cycle after a beat of normal length is more characteristic of auricular fibrillation than is a sudden shortening of the cycle

The customary *treatment* for patients with auricular fibrillation is digitalis The purpose is to slow the ventricular rate and if this slowing does not result from adequate dosage, one may rightly suspect the drug is not sufficiently potent or that the patient has hyperthyroidism Ordinarily the slowing is marked and may be compared in its specific effect to the action of quinine in malaria Furthermore, no similar slowing can be expected from digitalis on a normal tachycardia This explains why some cardiac patients improve after fibrillation develops, for the digitalis that had been given previously without effect only then begins to slow the rate

Quinidine on the other hand, is used at times to treat patients with auricular fibrillation (See Chap 20) It is used less than formerly but occasionally is a very valuable aid to our treatment The purpose here is to restore the heart to a regular rhythm (Figs 79 80) It does so by lengthening the refractory period of the auricular musculature so that when the wave completes its circuit it finds the tissue ahead of it still refractory and the circus ends, permitting the normal pacemaker to send out its impulse and reestablish ascendency over the beat of the heart The reason that

quinidine does not always break up fibrillation is that it also slows the speed of the impulse, which would tend to perpetuate the circus motion by allowing sufficient time for tissue to recover from its refractoriness When the first effect predominates fibrillation ceases, when the latter predominates it continues Quinidine is particularly useful in restoring normal rhythm if there is no organic heart disease and in a few other selected cases of auricular fibrillation

Ectopic Auriculoventricular (Nodal) Beats

Abnormal beats may arise in the auriculoventricular node or in the bundle of His The pacemaker in the junctional tissue may control the rhythm of the heart for long periods of time (Figs 83, 84) or only isolated beats may arise (Fig 86) The impulse will travel down the ventricles through normal pathways and therefore the ventricular complexes will be normal It reaches the auricles, however, in a reversed direction and so will produce abnormal P waves The auricular contraction may take place shortly before or after the ventricular, depending on which receives the impulse first If

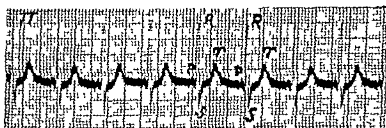


Fig 83 Nodal rhythm Note that the P waves have a peculiar form and that the P R interval is very short (0 06 second) The ventricular complexes are normal This patient had acute rheumatic carditis

the focus is near the top or auricular end of the node, it will reach the auricles quickly, but the P-R interval will be shorter than normal If the focus is lower down, the ventricles will respond before the auricles and an R-P rather than a P-R interval results (Fig 84) At times within the same tracing varying positions of the P wave will be found, sometimes before or after or even simultaneously with the R wave At other times the origin of the beat may vary in different parts of the sino auricular node and also in the auriculoventricular node, or one node may compete with the other in controlling the beat This condition is called a wandering pacemaker and will display different shapes of P waves (Fig 85) The normal beat from the sino-auricular node may produce auricular contraction before a nodal impulse reaches the auricles In this case the regular sequence of the P waves remains undisturbed although the premature nodal beat occurred (Fig 86) The auriculoventricular node often escapes whenever there is a long pause of the normal pacemaker, because it has its own irritability and rhythmicity which is then permitted to manifest itself

Nodal rhythms have no great clinical significance and cannot be identified without graphic methods They may occur during anesthesia, deep

breathing or vagal stimulation and can be produced by certain drugs There are no practical diagnostic, prognostic or therapeutic problems involved in this type of irregularity

Although nodal beats cannot be accurately identified without graphic methods, when a series of such beats arise and the relation of auricular and

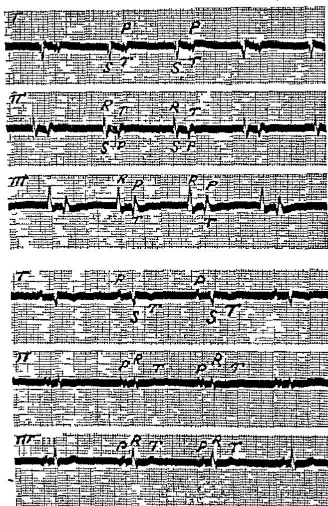


Fig 84 Nodal rhythm The upper three leads show P waves of a peculiar form coming at a constant interval after the R waves (R P interval of 0 23 second) The lower three leads show a return to the normal rhythm with a P R interval of 0 22 second The patient had alcoholic cirrhosis of liver

ventricular systole keeps changing one may suspect the presence of this type of arrhythmia by detecting a changing quality or intensity of the first heart sound This alteration is the result of the differences in the position of the auriculoventricular valves at the moment ventricular systole occurs A similar mechanism accounts for variations in the quality of the first heart

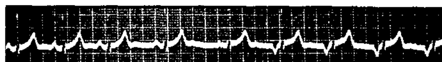


Fig 85 Wandering pacemaker The first four beats arise in the sino auricular node the last four in the auriculoventricular node The intervening (fifth) beat shows a flat P wave representing composite activation of the auricles from both the sino auricular and auriculoventricular nodes, so that the upper part of the auricles is stimulated from the sino auricular node and the lower part of the auricles from the auriculoventricular node This then is a combination or fusion auricular complex This sequence depicting migration of the pacemaker from one node to the other is one form of wandering pacemaker The pacemaker may also wander from the upper to the middle or to the lower part of the sino auricular or of the auriculoventricular node

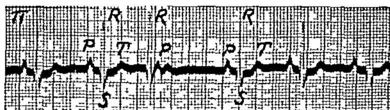


Fig 86 Premature nodal beat Note that the premature beat which arises in the auriculoventricular node produces a ventricular complex of normal form The auricular rhythm is undisturbed as the P wave comes exactly on time There was no heart disease here

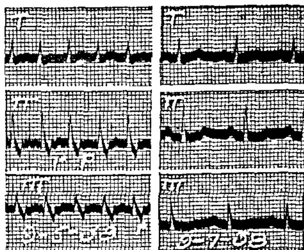


Fig 87 Paroxysmal nodal tachycardia (probable) The first set shows regular rate of 201 Note that P waves seem to occur at the R T junction The second set shows normal slow rhythm Some of these attacks were stopped by carotid sinus pressure The patient was a man 54 years old with tubercular constrictive pericarditis and obliterative pleuritis

sound in other arrhythmias, for this sound is mainly, if not entirely, due to the snap of the auriculoventricular valves

Paroxysmal Nodal Tachycardia

One would presuppose that a paroxysm of tachycardia might arise from the auriculoventricular node or junctional tissue just as it does from the auricles or ventricles It is difficult to be certain of this for when such a tachycardia displays abnormal P waves it cannot be determined whether such waves are the result of impulses arising in the auricles, in which case it really indicates paroxysmal auricular tachycardia or whether they come from the auriculoventricular node It is almost impossible to identify which ventricular complex is related to the particular P wave (Fig 87) In Figure 62, although at first sight this was regarded as paroxysmal nodal tachycardia, attention to the last beat in the paroxysm shows that the final ventricular beat in the paroxysm does not show an inverted P wave immediately after the QRS complex It is thus apparent that the inverted P waves following each of the other QRS complexes in the paroxysm is not retrograde from the ventricles but rather is conducted after a long P R interval (0 24 second) to the ventricles as the succeeding QRS complex From a clinical point of view whatever has been said about paroxysmal auricular tachcardia applies to the condition that might be called paroxysmal nodal tachycardia

Ectopic Ventricular Beats

Extrasystoles arising in some ectopic focus in the ventricle are extremely common (Figs 88 to 95) This arrhythmia is generally the cause of what



Fig 88 Premature ventricular beats probably arising from the left ventricle The normal rhythm is occasionally interrupted by ventricular complexes having a decidedly abnormal form (V) Note that the regularity of the auricular beats (P) is not interrupted although some are buried with the extrasystole The patient had neurocirculatory asthenia

the physician calls an intermittent pulse This latter expression should be given up because intermittence of the peripheral pulse can be due to a variety of disturbances of the heart beat, which can be properly diagnosed on auscultation of the heart In this condition the heart is beating regularly,

when suddenly a quick beat is heard followed by a pause The premature beat is the extrasystole and may occur so early in the previous diastole that the pressure and volume of blood in the ventricles often are not great enough to produce a pulse at the wrist (Fig 93) and occasionally may not even open the aortic valves In the latter case only one heart sound may be heard over the precordium for that particular beat I have even observed instances in which ventricular extrasystoles would be seen to occur while electrocardiograms were being taken which were entirely inaudible during auscultation that was performed simultaneously

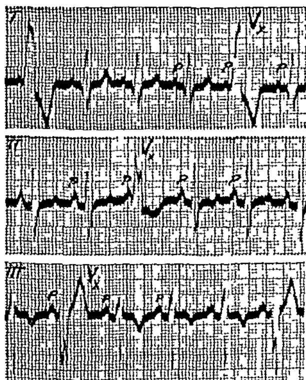


Fig 89 Premature ventricular beats probably arising from the right ventricle Note the extrasystoles with abnormal ventricular complexes (V_x) They are only very slightly premature so that they actually follow the P wave This patient had congenital heart disease

The spread of the impulse from the ectopic focus through the ventricle is abnormal and so the ventricular complex will be bizarre (Figs 88 to 95) The direction the waves will take will depend on whether the impulse arises in the left or right ventricle and in the basal or apical region In general the complexes are broad, coarsely notched and the T waves extend in the opposite direction to the main initial ventricular deflections Inasmuch as the ventricle contracts prematurely, the normal auricular beat coming down about the same time finds the ventricle refractory so that it cannot respond again There follows then a pause until the next normal auricular beat This pause is completely compensatory for the rhythm of

the pacemaker is not disturbed and the duration of the two beats including the extrasystole is equal to two normal heart cycles (Fig 90, Lead II) The

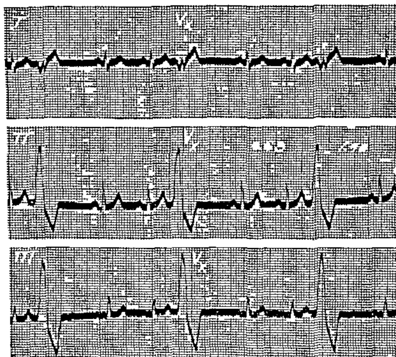


Fig 90 Premature ventricular beats Trigeminy Note the typical ventricular extrasystoles (V) coming regularly after two normal beats This patient had no organic heart disease The length of normal cycle is 0 69 second The length of the two beats including the extrasystole is just twice the normal beat 1 40 seconds Therefore the pause is completely compensatory

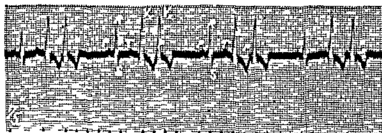


Fig 91 Premature ventricular beats Trigeminy Note that after each normal beat there are two consecutive extrasystoles $(V_x)_1$ followed by a compensatory pause The auricles (P) continue regularly The patient had rheumatic valvular disease (Author s article in Oxford Loose Leaf Medicine vol II)

P wave is often lost in the ventricular complex (Fig 90) or may appear before or after the QRS waves (Figs 88 and 89)

These beats may occur very rarely so that the physician is unable to detect them at the time of his examination or they may be frequent They

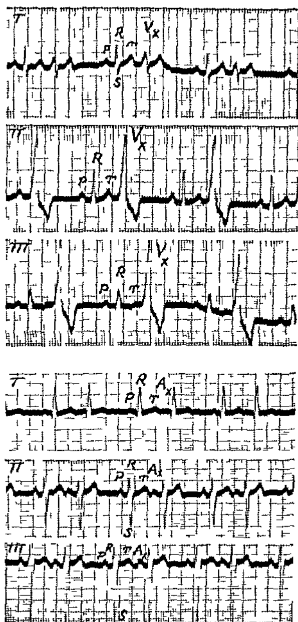


Fig 92 Premature ventricular beats Bigeminy The upper three leads show ventricular extrasystole (V_x) occurring after each normal beat and the lower hree show an auricular extrasystole (A_x) producing in both cases a coupled hythm Note the abnormal form of the ventricula complexes above and the ormal ventricular complexes below with auricular waves of abnormal form)rganic heart disea e was present in neither case

nay come every second beat (bigeminy) (Fig 92) or every third beat trigeminy) (Fig 90) Sometimes they come in succession so that there nay be a run of two or more extrasystoles producing curious types of

irregularity (Fig 91) When they arise in different parts of the ventricles they will have various forms (Fig 95) Such a condition is apt to be associated with serious heart disease

Occasionally ventricular extrasystoles are interpolated between two normal beats The heart is beating so slowly and the premature beat occurs so early in diastole that the next normal auricular impulse finds the con duction tissue and the ventricles ready to respond (Fig 96) There is no compensatory pause and instead one hears three rapid beats, the second one of which is the extrasystole Another possible explanation of this mechanism is that the impulse from the ventricles travels through the junctional tissue to the auricles in a retrograde fashion and starts a new impulse at the normal pacemaker Occasionally the beat following the interpolated ventricular premature beat displays prolongation of the P R interval or of the QRS complex (ventricular aberration) and the interval

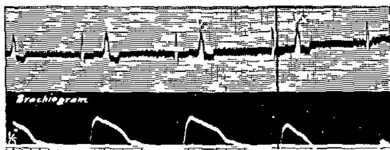


Fig 93 Premature ventricular beats bigeminy auricular fibrillation Every second beat is a ventricular extrasystole (V) Auricular fibrillation is present but the normal ventricular beats come regularly i e complete heart block Such curves result from toxic doses of digitalis Lower tracing is from the brachial artery and shows that the extra beats do not reach the wrist Apex rate 60 pulse rate 30 (Author s article in Oxford Loose Leaf Medicine vol II)

between the beat before and the one after the extrasystole may be longer than the regular beats (Fig 96)

Ventricular extrasystoles occur very frequently in individuals otherwise normal They are also common in those having organic heart disease but the diagnosis of structural disease of the heart will have to rest on other criteria than the irregularity When they are numerous and the patient requires digitalis for heart failure they indicate a grave outlook for it is more difficult to administer as large doses of the drug as would otherwise be possible In fact, digitalis in large doses often produces ventricular extrasystoles, especially in the form of digitalis coupling (Figs 93, 94), even if none were present before the drug was used Coupling may occur whether the rhythm was previously normal, in which case the P-R interval may also be delayed (Fig 94) or if auricular fibrillation was present (Fig 93) When this occurs it indicates a toxic effect of digitalis and the drug must be omitted or the dose should be diminished Coupling due to extrasystoles however, may occur without digitalis and even in the absence of heart disease

Ordinarily it is not difficult to recognize extrasystoles at the bedside When they are numerous they may resemble auricular fibrillation but they may generally be differentiated by the method discussed in a previous

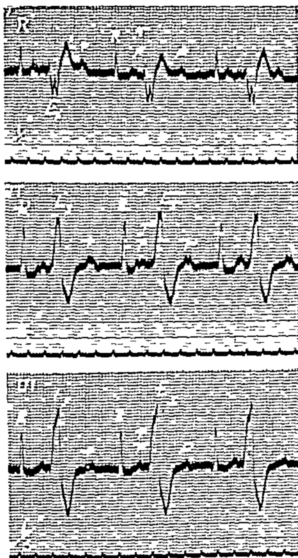


Fig 94 Digitalis coupling Every second beat is a ventricular premature beat with the typical abnormal form The auriculoventricular conduction time or P R interval is markedly delayed, measuring 0 42 second Both of these effects are typical of digitalis intoxication Note the cupped depressed RS T segments of the normal complexes typical of the effect of digitalis (Author s article in Oxford Loose Leaf Medicine vol II)

paragraph It is regarded as impossible to distinguish auricular from ventricular premature beats without graphic methods One may obtain certain suspicions that help to identify the one from the other If the observer taps with his foot in rhythm with the regular beats, and continues the same pace

when the extrasystole occurs the foot will come down synchronously with the following beat if it is a ventricular extrasystole This will generally not be so if it is auricular because the post-extrasystolic pause is not completely compensatory Furthermore the character of the sounds is different in the two types An auricular extrasystole sounds more like the normal beat, only coming prematurely A ventricular extrasystole has a peculiar clicking sound which is different from the normal first heart sound of the particular patient, because the ventricles are apt to be contracting simultaneously with the auricles or at least in an abnormal relationship to auricular systole This results in another distinguishing sign that may be elicited The fact that the auricles contract while the ventricles are in systole makes the auricular impulse in the jugular pulse more prominent and one may see a large a wave in the jugular vein This takes place because the blood from the right auricle is prevented from going into the right ventricle and is forced back through the superior vena cava However, when it is impor-

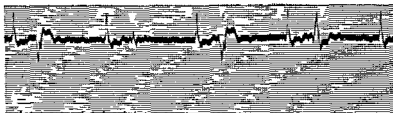


FIG 95 Multiple and multifocal premature ventricular beats Following each normal beat (R) there are ventricular premature beats having varying forms (V V_y V_z) These beats arise in different foci in the ventricles or have different conduction paths Such beats serve as a very important clue to digitalis over dosage but may at times be recorded in other conditions such as myocardial infarction (Author s article in Oxford Loose Leaf Medicine vol II)

tant to identify the type of extrasystole with certainty graphic methods will be necessary

The *main symptom* that is produced by ventricular extrasystoles is palpitation This will be variously described by different individuals and generally the diagnosis can be made from the history itself The various expressions used are skipping of the heart a sudden flop or hesitation, the heart flutters for an instant and then stops, a thump in the neck or chest (probably due to the beat after the pause which is always a vigorous contraction), a sudden sinking or faint sensation or a wave, and other terms often very graphic in their description Occasionally a momentary darting pain occurs with each extrasystole In many individuals they produce no symptoms and then are accidentally found on examination This is more commonly the case in stout patients and in those who are more phlegmatic It seems that with a thin chest the somatic disturbances within the chest cavity are more readily felt They are most frequent and most troublesome at rest, especially while a patient is trying to fall asleep and often are entirely absent during physical activity This may be due to the fact that as the

heart slows with rest, the diastolic pause lengthens and there is a greater opportunity for a premature beat to arise during longer than during shorter pauses There are other neurogenic factors that determine the development of these beats at one time or another apart from the heart rate There is no doubt that emotional disturbances can be responsible for their presence The following experience is quite illustrative This man who had the typical complaint of skipping of his heart did not happen to show any irregularity during my examination The symptom first began after the death of his child nine months before While his electrocardiogram was being taken and the heart was beating regularly I asked him what illness his child had, and as he began to speak about his child the extrasystoles appeared This fits in very well with the physiologic work showing that there is a center in the hypothalamic region which controls ventricular extrasystoles

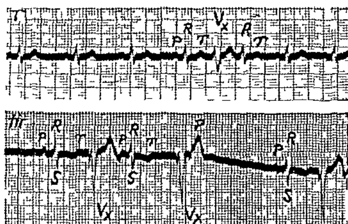


Fig 96 Interpolated premature ventricular beats Upper tracing shows a single ventricular extrasystole (V_x) interpolated between two normal beats Lower curve shows both an interpolated ventricular extrasystole and an extrasystole followed by the customary compensatory pause The first patient had no heart disease the second had angina pectoris

There is another type of ventricular extrasystole that only develops on physical effort In general this is more serious and may even predispose to the development of ventricular tachycardia on effort Occasionally in certain individuals they are produced by coffee or smoking More often when this is thought to be cause and effect, carefully controlled observation will show that there is no such relationship Often no known cause can be detected for their presence

As to *treatment*, it is best for the physician to regard extrasystoles lightly The patient should be clearly told that they have no serious significance and do not mean heart disease Some persons have them all their lives and are not handicapped thereby Such individuals should not be restricted in their activities unless there is some other reason for such restriction When extrasystoles produce no symptoms no medication should be advised When they are troublesome quinidine sulfate 0 2 to 0 3 gm (3 to 5 grains) two or three times a day or taken just before they usually occur may elim-

inate them entirely Occasionally moderate doses of digitalis, curiously enough, also inhibit extrasystoles Strychnine (0 001 gm —$\frac{1}{60}$ grain—three times a day) has also been recommended In some cases I have found that the extrasystoles disappear when the patient is given 2 0 gm (30 grains) of potassium phosphate or chloride three times a day Atropine sulfate may diminish vagal tone and increase the heart rate sufficiently to prevent their development In general it may be said that when it is important to eliminate extrasystoles, one or another of the various drugs that are available will prove effective in most cases

Paroxysmal Ventricular Tachycardia

An ectopic ventricular rhythm of still higher grade is paroxysmal ventricular tachycardia This may be regarded as a consecutive series of ventricular extrasystoles arising from an ectopic focus in the ventricle There is much in the nature of this mechanism that resembles a circus motion and it may eventually be proved to be a disturbance in the ventricles similar to flutter in the auricles Starting as it does from an abnormal focus in the ventricles, the impulse travels an abnormal course and the resultant ventricular complex will be abnormal (Figs 97 to 100) Each individual complex resembles a ventricular extrasystole The impulse may travel in a retrograde fashion up the junctional tissue and produce auricular contractions Because the rate is rapid, there may be a retrograde block so that only every other ventricular impulse reaches the auricles At other times the auricles contract independently and follow their own pacemaker in the sino-auricular node It is often impossible to identify the P waves because they are buried in the QRS-T complexes

It may be difficult to distinguish ventricular tachycardia from a rapid auricular rate with a block in one or the other bundle of His (Fig 99) Both conditions will show similar ventricular complexes The finding of isolated ventricular extrasystoles while the heart is beating slowly which resemble the complexes when the rate is rapid or the identification of P waves that are not in the normal relationship to the ventricular complexes will prove that the condition is ventricular tachycardia Figure 99 is an example of auricular flutter with bundle branch block which at first sight resembled paroxysmal ventricular tachycardia In this case the differentiation was relatively simple The problem may be more difficult with auricular fibrillation and a rapid ventricular rate combined with intraventricular block since the rhythm is irregular in either auricular fibrillation or ventricular tachycardia In general, it may be said that the irregularity is much more obvious and more easily detected in auricular fibrillation than in ventricular tachycardia In ventricular tachycardia the irregularity must generally be deliberately sought

For some hours or days after any attack of paroxysmal rapid heart action the ventricular complexes may remain distinctly abnormal showing inverted T waves These changes may occasionally occur when the heart is structurally normal and therefore cannot necessarily serve as evidence of heart disease They probably indicate heart muscle fatigue or local relative anoxemia of parts of the ventricles, but the curves return to normal even

tually Lack of appreciation of this will lead to incorrect diagnoses of coronary or myocardial disease

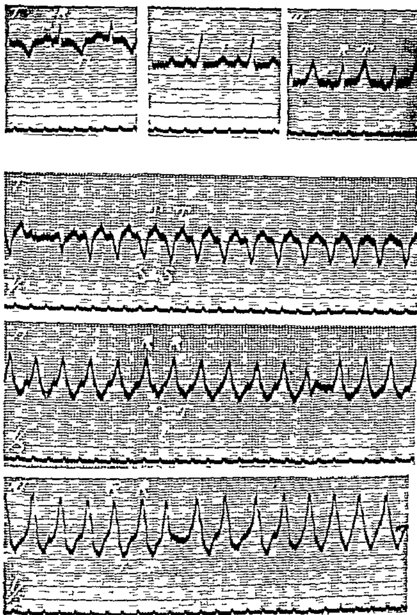


Fig 97 Paroxysmal ventricular tachycardia The upper three leads show a normal rhythm rate 89 The lower three leads were taken the next day during an attack of tachycardia rate 172 Note that the form of the ventricular complexes (RST) *changed markedly in the corresponding leads* There are slight irregularities in the length of the heart cycles during the tachycardia (Author s article in Oxford Loose Leaf Medicine vol II)

There are numerous important clinical aspects of paroxysmal ventricular tachycardia and this disturbance needs to be differentiated from other forms of rapid heart action It can occur as paroxysms or it may remain permanent

if treatment is not instituted The rate is apt to be around 160 to 180 and very rarely reaches the high levels of 220 or more that occur in auricular tachycardia Unlike the latter it generally accompanies grave heart disease, especially disease of the coronary arteries, but it may be present as a purely functional arrhythmia in an otherwise healthy heart Furthermore, there are bedside methods that enable one to diagnose this condition When paroxysmal, the attacks begin and end suddenly (Fig 98) The rate is rapid but in most cases slight irregularities can be detected on auscultation in marked contrast to auricular tachycardia (Fig 98) Occasionally the rhythm will be perfectly regular but more often, if one listens long enough, inter-

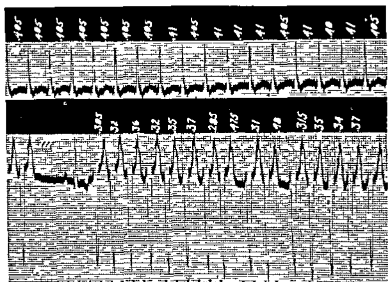


Fig 98 Paroxysmal ventricular tachycardia The upper tracing was made during an attack of auricular tachycardia the lower one shows the onset of ventricular tachycardia occurring in the same patient The numbers indicate the length of the heart cycles Note the absolute regularity of the auricular tachycardia and the distinct irregularity of the ventricular tachycardia

ruptions will be heard The first nine cycles in Figure 100 are just as regular as the cycles that occur in auricular tachycardia but thereafter appreciable differences in the length of the cycles develop that will never be seen in association with the latter condition Furthermore, on auscultation slight but definite differences in the intensity and quality of the first heart sound will be heard, which are due to the different relationship between the ventricular and auricular systoles in various cycles This latter auscultatory finding will not be present either in normal tachycardia or in paroxysmal tachycardia It also has been pointed out that the auricular pulsations as seen in the jugular vein will be fewer in number than the ventricular rate and some of these jugular pulsations are particularly conspicuous Finally, this type of rapid heart action is never influenced by any of the methods used to stimulate the vagus such as carotid pressure, ocular pressure or deep breathing

Therapeutically the problem of ventricular tachycardia is peculiar In most cardiac conditions in which there is heart failure digitalis is indicated and a beneficial, or at least not harmful, effect is anticipated When this

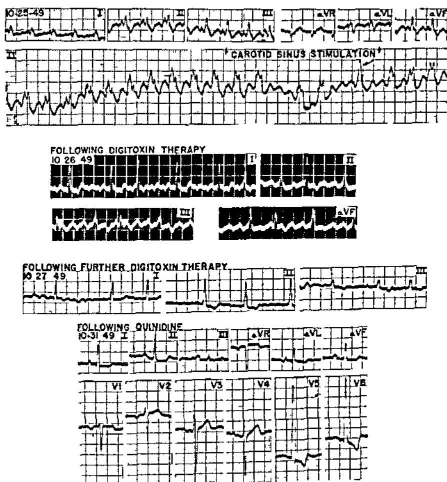


Fig 99 Auricular flutter with bundle branch block simulating paroxysmal ventricular tachycardia Initial tracings suggested paroxysmal ventricular tachycardia but the smooth hump between ventricular complexes suggested auricular flutter in the background Carotid sinus stimulation (Lead II) slowed the ventricular rate disclosing continuous flutter waves Following one long pause during induced slowing normal intraventricular conduction (see arrow) was produced indicating that the intraventricular block was a fatigue phenomenon which would be eliminated on slowing the ventricles Following digitoxin therapy auricular flutter with slower and varying ventricular response and as anticipated normal intraventricular conduction were produced After further digitoxin therapy auricular fibrillation occurred Following quinidine therapy normal sinus rhythm with ventricular complexes characteristic of left ventricular hypertrophy was produced

arrhythmia is present, digitalis will not only fail to improve the situation but may well worsen it I have seen two instances in which digitalis, on repeated trials, accelerated the ventricular rate while the tachycardia was in progress and aggravated the state of the circulation Quinidine, on the

other hand, is almost a specific drug for this type of tachycardia In most cases it will restore the normal rhythm The amount necessary to accomplish this will vary greatly from a single dose of 0 3 gm (5 grains) to as much as 1 5 gm (22 grains) administered five times a day Before the heart becomes regularized the ventricular rate will gradually slow (Fig 101)

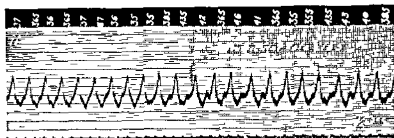


Fig 100 Paroxysmal ventricular tachycardia Numbers above indicate the inter ventricular intervals in hundredths of a second Note that although the first nine cycles are quite regular distinct variations in rate occur thereafter

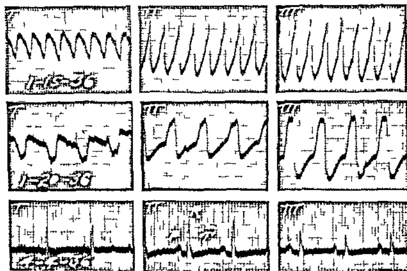


Fig 101 Paroxysmal ventricular tachycardia—quinidine effect First set shows ventricular tachycardia the rate is 247 Second set shows marked slowing of the rate (122) as a result of quinidine but ventricular tachycardia persists Lowest tracing shows normal rhythm Enormous amounts of quinidine were necessary to produce regularization single doses being gradually increased from 0 2 gm to 2 0 gm The patient was desperately ill with advanced congestive failure although after recovery there was no evidence of heart disease

This slowing enables one to follow the effect of the drug and guide its dosage On rare occasions even large doses of quinidine will only slow the ventricular rate but fail to do away with the abnormal mechanism, the original rapid rate returning as the effect of the drug wears off On two such occasions 2 mg ($\frac{1}{30}$ grain) of atropine sulfate, given subcutaneously

about one hour after a large oral dose of quinidine, promptly restored the normal rhythm The pharmacologic action of atropine is to lengthen the refractory period of cardiac muscle Also, 15 cc of 20 per cent solution of magnesium sulfate intravenously has been used successfully Quite recently

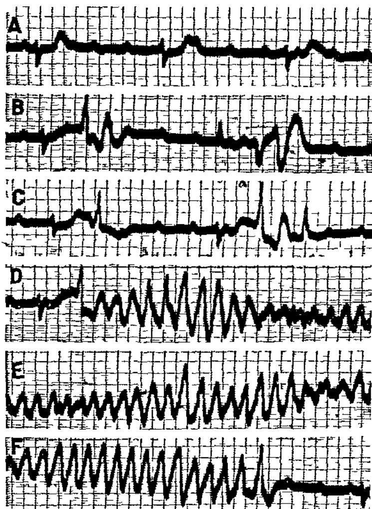


FIG 102 Ventricular flutter (possible) Strips of a continuous tracing (Lead I) from a woman 59 years old with complete heart block and Adams Stokes disease *A* indicates complete block *B* and *C* show ventricular extrasystoles of different types *D*, *E* and *F* show oscillations that have been interpreted as ventricular fibrillation but because heart beats were audible throughout this period it is suggested that it be called ventricular flutter The last complex shows return of complete block

procaine amide, 1 0 gm in 10 cc solution given intravenously, has been very effective and appears to have no serious side effects

Ventricular Flutter?

If one continues the sequence of abnormalities of ventricular origin, as was done in the auricles, one should naturally consider ventricular flutter

This generally has been omitted, as no condition has been recognized as ventricular flutter in the electrocardiograms Figure 102, however, may possibly represent such a condition These rapid, slightly irregular oscillations were taking place at the very time that very rapid heart beats could be heard The patient was unconscious However, she recovered from this attack Ordinarily one has considered such curves as signifying ventricular

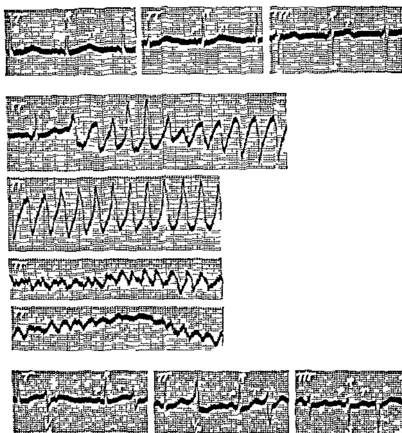


FIG 103 Transient ventricular fibrillation The upper three leads show a normal mechanism The following four curves are portions of a continuous tracing taken during an attack of syncope and are characteristic of ventricular fibrillation The patient had a convulsion and there was no heart beat or pulse for about one minute The lowest three leads show a return to a normal beat with occasional ventricular extrasystoles (V_x) The patient became ambulatory

fibrillation, but the presence of heart beats and the fact that the circulation was being maintained militate against this diagnosis and denote that the ventricles were actually contracting

Ventricular Fibrillation

The final and most extreme disturbance of the ventricles is ventricular fibrillation With this there are numerous impulses traversing the ventricles

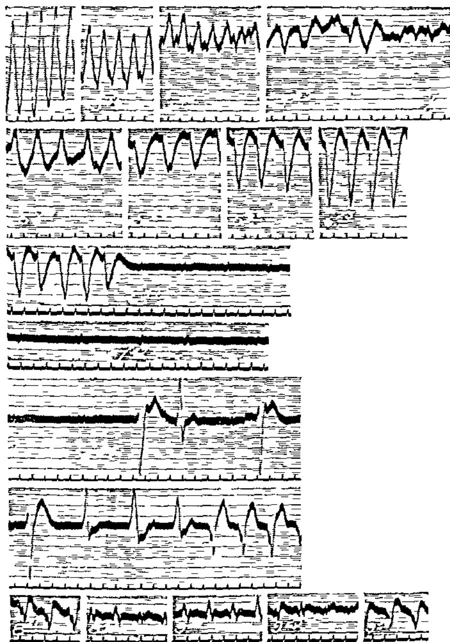


Fig 104 Ventricular fibrillation Portions of a continuous tracing taken during an attack of Adams Stokes syncope lasting five minutes The numbers indicate minutes and seconds The patient was unconscious throughout the attack breathing ceased and there was no detectable heart beat Note the early development of ventricular fibrillation with eventual cessation of all electrical activity except for small auricular waves (P) During this time adrenalin was injected directly into the heart and contractions were resumed The patient recovered and became ambulatory (Published in Heart vol. 12)

so rapidly that coordinated contractions do not occur When the condition is reproduced experimentally by faradizing the ventricles, tying the

branches of the coronary arteries or by drugs such as digitalis and adrenalin, fibrillary twitchings will be seen but no mechanical expulsion of blood results From the point of view of the dynamics of the circulation the heart suddenly stops The electrocardiograms that represent this state show very bizarre rapid and irregular ventricular complexes (*Figs 103, 104*) The initial phase of the ventricular waves (QRS) is broadened and the T waves fuse with them so that they become indistinguishable They also vary in height from oscillations of large amplitude to coarse low movements of the base line

It is extremely rare to find ventricular fibrillation in clinical practice because in most cases it is the cause of instant death By mere chance, examples have been recorded when a patient happened to have an attack while the electrocardiograms were being taken This was the case in an instance of sudden death from an attack of angina pectoris in which the

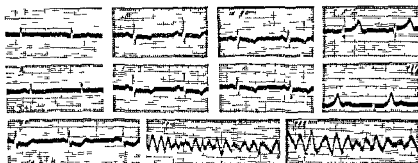


Fig 105 Ventricular fibrillation (cause of sudden death in coronary disease) This patient a man 60 years old had typical angina for three weeks with many attacks daily promptly relieved by nitroglycerin On the last day he had a severe attack requiring two injections of morphine and died instantly two hours after the onset of the last spell while tracings were being taken Note the normal rhythm in Lead I of lowest set and ventricular fibrillation in Leads II and III taken only a few seconds later

postmortem examination showed disease of the coronary arteries but no acute thrombosis This mechanism probably is a common cause of instant death in disease of the coronary arteries and from such causes as electrocution Figure 105 shows the recording made when a patient happened to have an attack while electrocardiograms were being made This patient had previous angina and had had a recent acute myocardial infarct He was doing quite well and just after the first lead of the electrocardiogram was taken he expired Leads II and III, taken only a few seconds afterwards, showed that ventricular fibrillation was present

Occasionally recovery takes place in human beings after ventricular fibrillation has set in Figure 103 shows an example of this sort, in which repeated attacks of syncope were due to this mechanism and were generally preceded by brief periods of increased numbers of ventricular extrasystoles and ventricular tachycardia The number of attacks seemed to be lessened by constant quinidine administration In Figure 104 are shown not only

periods of ventricular fibrillation but also complete cessation of all electrical responses in the ventricles, extending over a period of more than five minutes, with recovery Whether the adrenalin that was injected directly into the heart was responsible for the recovery is not certain This occurred in a woman about 50 years of age who was suffering from Adams-Stokes disease with attacks of unconsciousness

DISTURBANCES IN CONDUCTION

Sino-auricular Block

Having considered the disturbances that may arise in the normal pacemaker of the heart and then the abnormalities in the origin of impulses (ectopic rhythms), the third main problem is to discuss the abnormalities

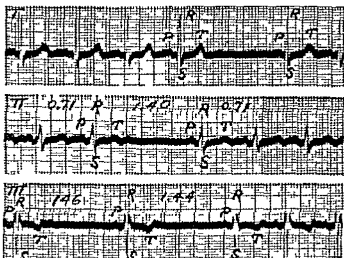


Fig 106 Sino-auricular block. Note that the pauses (1 4 seconds) are equal to twice the length of the normal beat (0 71 second) Neither auricles nor ventricles contract during the pauses as the impulse is blocked at the pacemaker The patient was a young woman with palpitation but without organic heart disease

in the conduction of impulses The impulse after it is formed in the normal sino-auricular node may be blocked before it reaches the auricular musculature This is called *sino-auricular block* (Figs 106, 107) Neither the auricles nor ventricles receive an impulse and there results the loss of a complete heart cycle If only one of those normal impulses is blocked, the pause will be approximately equal to two normal heart beats as the pacemaker remains undisturbed Occasionally this pause will be equal to three or four heart beats in which case two or three impulses are blocked Slight differences in measurements will be found because there often is an accompanying sinus arrhythmia that slightly affects the pace Furthermore, after the pause the first beat may be an idioventricular beat or nodal escape

Sino-auricular block is rare compared to auriculoventricular block In general it occurs under the same conditions but has much less practical

importance It can be suspected clinically by detecting a complete loss of a heart cycle on auscultation Because neither auricles nor ventricles contract during this pause, no auricular or a wave will be seen in the jugular pulse If such a small wave is detected on careful examination of the veins of the neck during the long silent interval, the block is at the auriculoventricular node rather than at the sino auricular node Treatment is generally not indicated for patients with this arrhythmia unless it produces

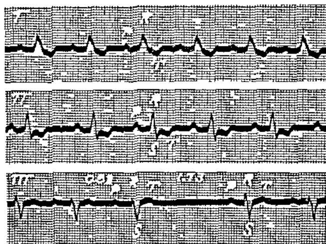


Fig 107 Sino auricular block There is a pause in Lead III (1 73 seconds) approximately twice the normal heart cycle (0 92 second) Both auricles and ventricles failed to contract Curves also show bundle branch block and delayed conduction time (P R = 0 27) The patient had hypertensive heart disease with coronary artery sclerosis

syncope when ephedrine or atropine may prove effective as in the ordinary type of block

Auriculoventricular Block

First Degree Heart Block (Delayed Conduction Time) The more common type of heart block is that which occurs in the junctional tissue at the auriculoventricular node or bundle of His When the defect is slight, impulses are merely delayed in their passage through the main conduction path but eventually reach the ventricles There really is no blocking of beats and the heart remains regular The conduction time (P R interval) which normally measures about 0 16 second becomes prolonged and when it reaches the duration of more than 0 2 second it is arbitrarily regarded as an indication of pathologic change (Figs 108 to 112) The actual path that the impulse travels is normal and therefore the electrical complexes are all normal in form The P wave may precede the ventricular complexes sufficiently to fall on the previous T or R wave Under such circumstances it may become difficult to recognize the P wave and it may confuse the interpretation of the electrocardiograms At first glance the tracings in Figure

112 as represented by the first three beats could be interpreted as paroxysmal tachycardia (rate 162) in which the auricular waves are not visible.

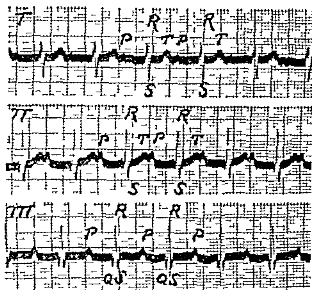


Fig 108 Heart block first degree (delayed conduction time) The P R interval measures the time it takes for an impulse to go from auricles to ventricles and normally should be between 0 12 and 0 2 second Here it measures 0 3 second None of the beats is blocked so that the heart remains regular The patient was suffering from postscarlatinal rheumatism

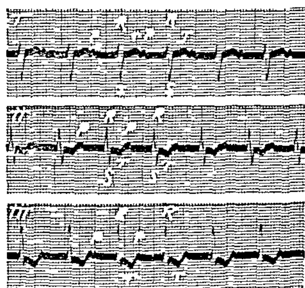


Fig 109 Heart block first degree The P R interval is markedly delayed measuring 0 32 second but no beats are blocked The patient had rheumatic mitral stenosis and insufficiency

Vagal stimulation, however, shows that the P waves are on top of the T waves and that there is a slightly delayed P-R interval

In general, auriculoventricular block of all degrees results from three main causes Digitalis probably is the most common of all When full doses of this drug are given any degree of heart block may be produced, especially delayed conduction (Figs 82 and 94) Even sino auricular block may be caused by digitalis Heart block rarely occurs during the ordinary infections met with in general practice such as scarlet fever or mumps It is more common as a complication of rheumatic fever and diphtheria When heart block of the higher grade occurs in diphtheria the prognosis is very grave In rheumatic fever heart block is particularly common and often is the only evidence of a rheumatic infection It can then be interpreted as posi tive evidence of a rheumatic carditis and may be present when there are



Fig 110 Heart block first degree The P R interval is delayed to 0 32 second but the rhythm remains regular Note that in Lead II the P wave is practically fused with the preceding T wave The patient was a boy of 13 who had an apparent cold the electrocardiographic changes being the only evidence at that time that the infection was rheumatic

no rheumatic pains and when the heart itself shows no significant abnor malities on ordinary examination except this electrocardiographic finding In fact, it is practically the only reliable evidence of an acute myocarditis Because of the infrequency of heart block in other acute infections, this finding should be sought for and will often serve as a valuable diagnostic aid in identifying many conditions as rheumatic that otherwise would remain obscure Figure 108 (P-R = 0 3 second) was obtained from a patient who had some vague aches in the legs after scarlet fever and was interpreted as indicative of a smouldering rheumatic fever Another patient (Fig 110) merely had a slight cold and a questionable murmur in the heart The markedly delayed conduction (P R = 0 32 second) meant that the child was suffering from a mild rheumatic fever More important still was the

case illustrated by Figure 111 Here a girl of 13 was spared an unnecessary operation for appendicitis by the interpretation of the abdominal pain, slight fever and leukocytosis as rheumatic The third major cause of conduction defects is chronic organic heart disease, especially coronary artery

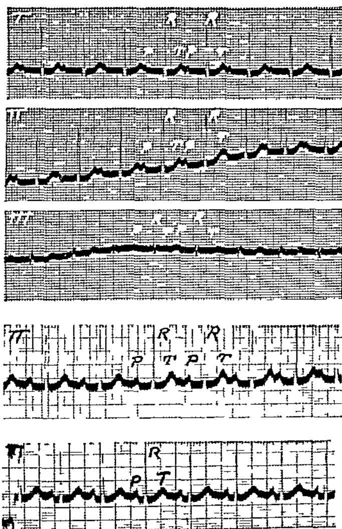


Fig 111 Heart block first degree The upper three leads taken June 20 1935 show delayed P R interval of 0 3 second The middle curve taken July 5 1935 shows that the P R time is much shortened 0 23 second The lower set taken September 5 1935 shows normal P R interval (0 16 second) The patient was a girl of 13 with vague abdominal ymptoms who was sent to the surgical service for appendectomy The electrocardiograms were the main clue that she had rheumatic fever

sclerosis and less frequently rheumatic valvular disease (Fig 109) Hyperthyroidism and syphilitic disease involving the junctional tissue are also rare causes of heart block When delayed conduction results from digitalis, it disappears within two or three weeks if the drug is omitted When rheu-

matic fever is the cause, it may persist longer but generally clears up entirely (Figs 111 and 114) However, when it is part of a chronic cardiac problem it remains indefinitely and may become more marked, eventually leading to complete heart block I once saw a man 59 years old who had fainting attacks with convulsions due to heart block On postmortem examination a lesion was formed in the upper portion of the interventricular septum resembling Fiedler's myocarditis There was no evidence of syphilis and no significant coronary artery disease In another somewhat similar case of Adams-Stokes disease the pathologist described lesions in the interventricular septum that were characteristic of sarcoid

It must be borne in mind that slight and occasionally marked delay in

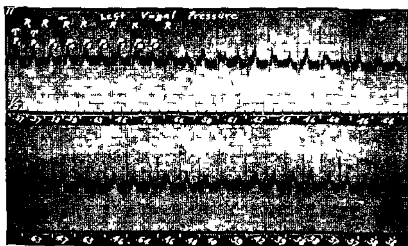


Fig 112 Heart block first degree Effect of vagal stimulation A continuous tracing at the beginning of which the heart was regular and rapid rate 162 The auricular waves were obscured and it was thought that paroxysmal auricular tachycardia was present Pressure over the left carotid sinus (indicated by signal above) slowed the ventricles and uncovered normal P waves Note that P_5 and P_8 are not followed by ventricular contractions The condition was an acute myocarditis following erysipelas with a normal sinus tachycardia and delayed conduction time (Author's article in Oxford Loose Leaf Medicine vol II)

auriculoventricular conduction may be found in otherwise healthy and normal individuals During the recent World War a P R interval of 0 4 sec was detected in some healthy Air Corps men while recumbent which became normal on sitting up In other instances second or third degree block was changed to first degree on assuming the upright position

Although first degree heart block is easily recognized in the electrocardiogram it is difficult and often impossible to detect it clinically The heart remains regular and the rate is often essentially normal Unless the possibility is kept in mind there may be no suspicion that a defect in conduction is present There are two auscultatory findings that may lead one to the correct diagnosis, i e, the presence of a gallop rhythm or a decrease in the intensity of the first heart sound It may be that auricular systole is

so far removed from the oncoming ventricular beat that it becomes audible or auricular contraction produces some other effect on the valves or muscle which results in a sound This finding should make the examiner particularly suspicious if it occurs during or following an acute infection Of course, gallop rhythm is much more common in other conditions in which the P-R interval is normal Nevertheless, it may direct attention to an important disturbance that otherwise would be overlooked Similarly if the loudness

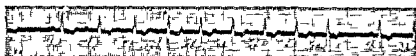


Fig 113 Heart block second degree or partial heart block Note that the conduction time (P R) first rapidly increases, then remains prolonged and finally a beat is blocked the process repeating itself The patient had advanced heart failure with hypertension and was on full digitalis dosage when these tracings were taken

of the first heart sound has been noted to decrease during the days of observation, it may mean that the P-R interval has lengthened

Second Degree Heart Block (Partial Heart Block) When the defect in conduction is greater, impulses from auricles to ventricles find it increasingly difficult to pass through the junctional tissue until finally one is blocked This condition is called *partial heart block* (Fig 113) This may occur once every seven or more beats or much more frequently, i e, every



Fig 114 Heart block second degree The upper tracing was taken June 25 1929 during acute rheumatic fever the lower October 20 1930 when the patient was well and showed no evidence of heart disease Note that P_3 and P_5 are not followed by ventricular beats The lower curves show that the heart block entirely disappeared

second beat (Figs 114, 115, 116) In fact, more than one beat may be blocked before the next one goes through These various degrees of partial block will be called 7 6, 2 1, or 3 1 respectively, depending on the relation of the P waves to the ventricular complexes The auricular and ventricular complexes are normal in form for that heart because the path which the impulses take is normal The P waves may have to be sought for carefully in a preceding T wave A slight notching or increase in height

of the latter may indicate the presence of the former (Fig 110) The time relation between the two will be abnormal Figure 113 shows a rapid in

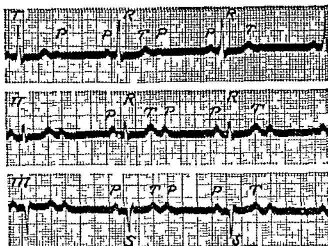


Fig 115 Heart block second degree Every other auricular beat (P) is not followed by a ventricular beat i e there is a 2 1 heart block Auricular rate 86 ventricular rate 43 The patient had hypertension angina pectoris but no syncope

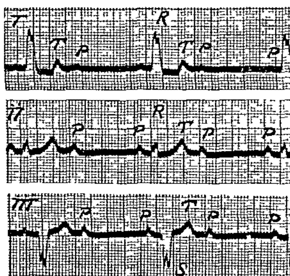


Fig 116 Heart block second degree Every other auricular beat (P) is blocked (2 1 heart block) Auricular rate 80 ventricular 40 The ventricular complexes (QRS T) indicate bundle branch block in addition Note that the P P interval embracing the ventricular complex is shorter than the clear P P interval The patient had hypertension and fainting attacks

crease in the P-R interval in the first four cycles and then the intervals remain the same until a beat is blocked The increase might have continued but to a slighter degree before the final pause Because of this diminishing

increment in the increase of the P-R interval the heart speeds up before the pause and the shortened P-R interval after the block makes the length of the pause slightly less than two normal heart cycles (Wenckebach's phenomenon)

It is not difficult to recognize partial heart block at the bedside When only an occasional beat is blocked an essentially regular rhythm will be heard and then a sudden pause that will be slightly shorter than two normal cycles A small auricular wave may be observed in the jugular veins during these pauses It must be clearly distinguished from extrasystoles for in both conditions the pulse is intermittent With the latter an extra clicking beat will be heard in early diastole On rare occasions I have confused this sound for the faint sound made by auricular systole in partial heart block When there is a 3 2 block a coupled rhythm results and this may be misinterpreted as due to some other mechanism If every second beat is blocked (2 1) a slow regular rate of about 40 is the result It will need to be distinguished from a normal bradycardia and complete heart block This often can be done by detecting auricular impulses in the jugular veins

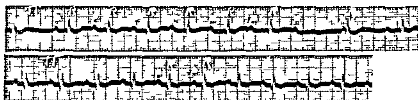


Fig 117 Second degree heart block relieved by atropine The upper tracing shows gradual lengthening of P R interval from 0 21 to 0 32 second and final blocking of a beat The lower curves taken five minutes after the intravenous injection of 1 0 mg of atropine show regular rhythm P R = 0 17 second

and trying to disturb the rate by exercise, breathing, carotid sinus pressure or drugs such as atropine or amyl nitrite The effect in normal bradycardia will be most marked and gradual, in complete heart block the rate will be unaffected or only slightly changed, the rhythm remaining regular, with partial block abrupt changes in the length of the heart cycle may result Atropine or amyl nitrite may lessen the degree of partial block by diminishing vagal tone, and vagal stimulation may increase the block and slow the heart further Occasionally the reverse occurs and ventricular rate accelerates with vagal stimulation In an elderly man with 2 1 partial heart block, carotid sinus stimulation on three different occasions promptly produced a 1 1 rhythm by slowing the auricular rate slightly There appeared to be a critical level at which all beats could be conducted and above which 2 1 block occurred This may explain the occasional beneficial effects of digitalis on heart block Careful attention to the physiologic functions involved and to the exact length of the heart cycles as determined by auscultation will often suffice to differentiate the cause of the slow heart rate

Partial heart block generally requires no specific treatment When it

accompanies rheumatic infections, treatment is directed at the underlying disease It merely indicates that a longer period of convalescence is desirable but the prognosis is ordinarily favorable If it follows digitalis, no harm results It serves as a warning that the dose of the drug should be lessened When it is present with chronic organic heart failure, one need not be afraid to give digitalis Specific treatment will be required if syncopal attacks are threatening This will be discussed under complete heart block One might expect that atropine would be of aid in partial heart block by inhibiting the vagus, and occasionally it is very effective in eliminating the block entirely and doing away with the accompanying symptoms (Fig 117)

Third Degree Heart Block (Complete Heart Block) The highest degree of block occurs when the defect is sufficiently great to prevent all impulses from reaching the ventricles, i e , complete heart block In this case the auricles contract in sequence to their own pacemaker in the sino-auricular

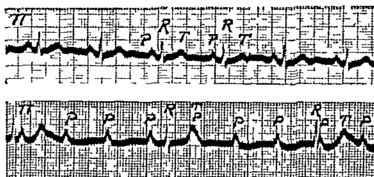


Fig 118 Heart block third degree (complete heart block) The upper tracing taken September 23 1931 shows a normal rhythm The lower tracing taken May 23 1933 shows complete block auricular rate (P) is 103 ventricular (R) is 29 Note the inconstant relationship between P and R waves The pacemaker of the ventricles is in the auriculoventricular junctional tissue The patient had angina pectoris and Adams Stokes disease

node and the ventricles establish their own rhythm in sequence to a pacemaker just below the defect either in the lower portion of the auriculoventricular node or in the bundle of His (Figs 118 to 124) Inasmuch as the inherent rhythmicity of the ventricles is slow, the rate is generally about 30 and regular The auricles and ventricles contract independently of each other Sometimes the P wave precedes the R, at other times it follows it or comes on the T wave Occasionally the auricular rate may happen to be just about twice the ventricular and an apparent 2 1 heart block will be seen However if a sufficiently long tracing is followed or an attempt made to disturb the mechanism, a P wave will generally be found to change its relation to the R wave and actually pass it The complexes have forms that are essentially normal for that particular heart as the spread of the excitation wave is normal through auricles and ventricles The Q-T interval is apt to be lengthened because of the slow rate and the increased duration of systole Although the ventricular rhythm is usually regular, slight irregu-

larities may be present Curiously, the ventricular contraction may disturb the auricular rhythm (Fig 116) possibly by changes in blood supply, through nervous reflexes or through the phenomenon of "synchronization"

Although the characteristic ventricular rate in complete block is very slow, rates of 40, 50 and more occur This is often true when the complete block is due to digitalis (Fig 82) Higher rates are also found when complete heart block is present in childhood (Fig 120) and in congenital

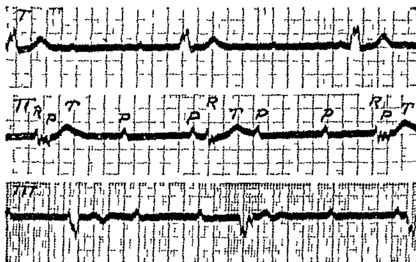


Fig 119 Complete heart block Auricular rate 66 ventricular 27 Note that the auricular waves (P) may come just before during or after the ventricular complexes (R T) The patient had rare attacks of Adams Stokes syncope but was able to do hard work The ventricular complexes are regular They could arise in the auriculoventricular node and be blocked in one of the bundle branches or they could arise as ectopic idioventricular beats in or near the opposite bundle branch In view of this uncertainty it is safer to refer to them with the noncommittal diagnosis of complete heart block with abnormal ventricular complexes

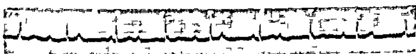


Fig 120 Complete heart block Auricular rate 90 ventricular 51 Note slight irregularity in the ventricular rate The patient was a boy 17 years old, with probable congenital ventricular septal defect and symptoms of cardiac weakness

heart block (Fig 121) The exact rate probably depends on the site of the idioventricular pacemaker When the lesion is higher in the auriculoventricular node, the rate will be faster In general, the lower portions of the heart have more sluggish rates It is also true that notwithstanding the fact that vagal influences do not affect the ventricles very much, the upper part of the auriculoventricular node is somewhat under the influence of the vagus, but this effect diminishes at the lower levels of the junctional tissue as the inherent rate decreases

The most important practical aspect of complete heart block is that there is a tendency to syncopal attacks accompanying it These attacks are due to further slowing of the ventricles or to complete ventricular standstill When the rate remains constant, even as low as 30, the efficiency of the circulation may be normal I have seen many instances in which patients were able to do hard physical work for many years with such slow hearts Danger arises when the ventricles stop contracting entirely Such pauses come suddenly and the symptoms they produce will depend on their length If the pause lasts several seconds the patients may only feel a faint wave or light headed sensation like a petit mal If it lasts a little longer, they faint away for several seconds If it continues for twenty to sixty seconds they will lose consciousness and have a convulsion with stertorous

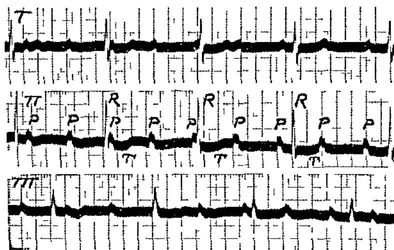


Fig 121 Complete heart block This patient was 55 years old known to have had heart block at least since the age of 6 probably congenital and never had any symptoms referable to the heart Note that the ventricular rate of 44 with congenital heart block is apt to be more rapid than with the acquired typ in adults (compare Figs 118 and 119)

breathing If it lasts a few minutes, breathing ceases and death generally ensues On very rare occasions the normal beat may be resumed even after the heart has ceased beating for several minutes (Fig 104) Attacks of syncope may occur at the sudden onset of the complete heart block before the new ventricular rhythm takes on its function They may also come at the time of the transition between a partial and complete block or when further depression of the pacemaker occurs and the rate falls below 20 (Fig 122) When, for some reason, possibly through changes in blood supply or nervous influences the ventricles which were contracting regularly at the rate of 30 or 20 suddenly stop entirely syncope will occur Finally, if a patient with complete block develops an attack of paroxysmal ventricular tachycardia it is apt to be followed by complete standstill of the ventricle (Fig 104) The precarious feature of complete heart block is,

therefore, not the slow regular rate of 28 but the temporary complete failure of the idioventricular pacemaker to send out impulses Similar syncopal attacks may occur even without complete auriculoventricular block if the ventricles fail to contract, as may take place reflexly from a sensitive carotid sinus or in sino-auricular block

The clinical recognition of complete block is ordinarily a simple matter



Fig 122 Complete heart block The auricles (P) are beating regularly at a rate of 111, the ventricles are contracting slowly and irregularly at a rate of about 16 The two rhythms are independent of each other (Author s article in Oxford Loose Leaf Medicine vol II)

when the rate is very slow and regular as there is no other condition in which this occurs When the rate is between 35 and 50 or more it can be confused with other conditions, especially partial block The differentiation has already been discussed in the paragraphs on partial heart block There is one additional sign that is pathognomonic of complete block, i e , the changing quality and intensity of the first heart sound On careful

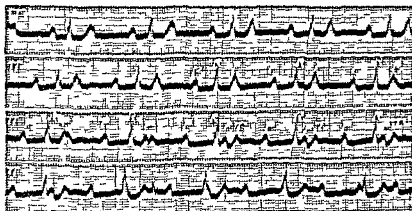


Fig 123 Complete heart block (false 2 1 block) Continuous tracing shows that while for a long period (upper two strips) a 2 1 relationship appears to exist complete block finally develops (lower two strips) with the P waves crossing the R waves

auscultation it will be noted that with occasional cycles the first heart sound may become muffled, accentuated or reduplicated This results from the changing relationship between the auricular and the ventricular contractions In some cases the auricular rate is exactly twice the ventricular for long periods of time so that only after prolonged auscultation will this valuable diagnostic sign become apparent (Fig 123) Furthermore, it is often possible to hear extra auricular sounds over the precordium during

the long pauses, to see extra auricular waves in the jugular pulse and even to feel faint waves in the radial pulse synchronously with auricular systole which are due to an impact against the aorta All these latter signs merely indicate that the auricles are contracting more frequently than the ventricles and that there is some degree of block The changing quality of the first heart sound and other criteria prove that the block is complete

The clinical conditions in which complete heart block occurs were taken up under partial heart block In some cases of otherwise inexplicable complete block the conduction defect appears to be related to a severe attack of diphtheria in childhood In many conditions, once established,

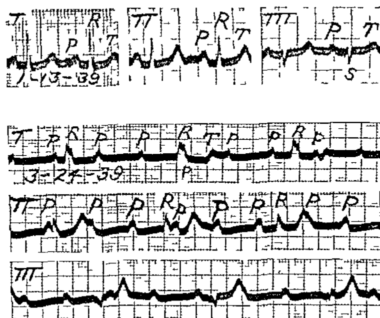


Fig 124 Complete heart block (Adams Stokes attacks with normal rhythm between spells) The patient was a woman 60 years old with hypertension and mitral stenosis At the time the first tracing was taken she already had had frequent attacks of unconsciousness Two months later she showed typical complete block and syncopal attacks continued

complete heart block is apt to be permanent, although occasional reversion to a block of lesser degree or a normal mechanism occurs Although mild degrees of heart block are common during the acute stages of rheumatic fever complete heart block is rare Furthermore, it is very uncommon to find permanent complete block as a sequel of rheumatic fever unless it develops many years later as an accompaniment of valvular disease

Although the majority of cases of heart block associated with attacks of Adams-Stokes syncope will show a slow heart and complete block between spells there is a considerable number of patients who have a normal rate and no block except when they are having attacks It follows, therefore,

that finding a normal rhythm in a patient with fainting spells by no means rules out the diagnosis of Adams-Stokes attacks (Fig 124) In Figure 125 is illustrated an unusual condition in which complete block is transient, changes to 2 1 block and is accompanied by probable bilateral bundle branch block

Therapy in complete heart block is important because it is often effective Treatment is not directed at the slow steady rate, for this many be productive of no symptoms and may even be a protection to a heart that is threatened with congestive failure However, the slow rate is no contraindication to the use of digitalis if congestive signs are present The main concern is the attacks of syncope When they occur at rare intervals, once every year or more, the problem is difficult because preventive medication would have to be continued all this time and it might even then be doubtful whether or not the cessation of attacks was due to the drugs When attacks occur frequently, many a day, as may happen during acute coronary thrombosis, adrenalin given subcutaneously may be life-saving It may be necessary to give a dose of 0 3 to 0 5 cc of a 1 1000 solution every two hours

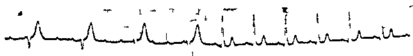


Fig 125 Complete heart block resulting from bilateral bundle branch block The patient was a 64 year old man with Adams Stokes attacks The auricular rate remained unchanged throughout the curve During the second half of the tracing there was second degree (2 1) auriculoventricular heart block with left bundle branch block during the first half of the tracing complete heart block with abnormal ventricular complexes resembling block in the opposite bundle branch The changes can be explained as the result of block in both bundle branches equivalent to complete heart block during the first half of the strip and of disappearance of block in the right branch during the second half

for a day or so The frequency and duration of the treatment will depend on the circumstances For a period of an hour or more after adrenalin is injected all attacks may cease The drug increases the irritability of the ventricles and not only prevents the pauses of the ventricles but actually increases their rate in complete heart block When the condition is chronic and the attacks occur in an otherwise ambulatory patient, ephedrine sulfate administered by mouth (0 025 gm —$\frac{3}{8}$ grain—or even larger doses) two or three times a day may prove effective Occasionally barium chloride (0 03 to 0 06 gm —½ to 1 grain—three or four times a day) given by mouth will inhibit all attacks In stubborn cases a great variety of procedures have been employed and when an apparently favorable result has been obtained it is difficult to be certain of the efficacy of the agents employed in treatment Amongst these are inhalations of 1 100 adrenalin, benzedrine, propadrine, full doses of atropine, intravenous injections of 50 per cent glucose, thyroid extract, cardiazol, and digitalis or urginin It must be remembered that quinidine should be given with caution to patients with disturbances in conduction as it can increase the inhibition of beats

CONGENITAL HEART DISEASE

In only one congenital condition, namely, true congenital dextrocardia, is the electrocardiogram absolutely pathognomonic The electrical axis of the heart lies in the direction opposite to normal, i e, from left to right When the ordinary three leads are taken, therefore, Lead I will be a mirror picture of the normal Lead I All the waves will be inverted (Fig 126, upper tracing and Fig 127) Lead II will resemble a normal Lead III and vice versa Similarly Lead aV$_R$ resembles normal Lead aV$_L$ and Lead aV$_L$ resembles normal Lead aV$_R$ Leads over the left precordium resemble those over the normal right precordium and vice versa (Fig 127) If the right and left arm electrodes are reversed curves of normal appearance will be obtained (Fig 126 lower tracing) Care must be taken in making the diagnosis of dextrocardia from electrocardiograms, because accidental interchange of the arm electrodes will produce similar curves This mistake is not at all uncommon

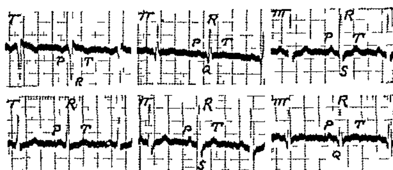


Fig 126 Dextrocardia The upper tracing shows the ordinary three leads in a case of congenital dextrocardia with situs inversus of the abdominal viscera Note that all the waves (P R T) are inverted in Lead I Leads II and III appear inter changed The lower curves were obtained on the same patient by placing the right arm electrode on the left arm and *vice versa* They then appear to be normal curves

In three other congenital conditions the electrocardiogram provides decisive evidence of the nature of the defect, if considered in the light of the clinical findings These are congenital complete heart block, congenital atresia of the tricuspid valve and congenital origin of the descending branch of the left coronary artery from the pulmonary artery The electrocardiographic clue to the diagnosis of congenital tricuspid atresia is the presence of left ventricular hypertrophy in a cyanotic infant In anomalous origin of the left coronary artery from the pulmonary artery electrocardiographic evidence of anterolateral ischemia has been reported Apart from these conditions it is a fairly valid general rule that extreme right axis deviation or extreme right ventricular hypertrophy indicates some form of congenital heart disease

The electrocardiographic examination must be regarded as a very important incident in the work up of every patient suspected of having a

congenital cardiovascular lesion The electrocardiogram parallels the anatomic and physiologic changes quite closely and records changes if the abnormal flow or the pressure changes are adequate to produce hypertrophy Thus, in a general way, the electrocardiogram corrects or confirms the roentgenographic and fluoroscopic findings regarding ventricular hypertrophy Indeed, the electrocardiogram is somewhat more reliable than x-ray examination in the detection of right ventricular hypertrophy While the x ray may detect ventricular enlargement in general, it is relatively inaccurate in telling which ventricle is enlarged In pulmonic stenosis

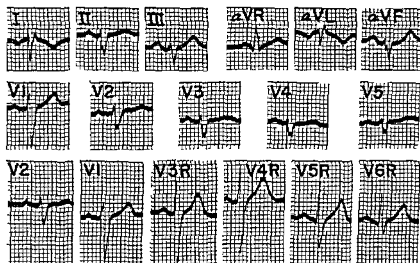


Fig 127 Dextrocardia studied with unipolar leads The conventional limb leads show the same type of change described in the previous figure (Fig 126) Note that Lead aV_L here resembles the normal Lead aV_R Lead aV_F has a normal relationship to the heart The second row of tracings shows Leads V_1 to V_5 taken in the usual manner Note that instead of increasing as the electrode moves to the left the R wave actually decreases By interchanging Leads V_1 and V_2 and continuing over the right side of the chest representing the true precordium, a series of tracings is obtained comparable to the usual set of precordial leads Note now that the R wave shows a progressive increase in height to Lead V_{5R} and therefore that when rearranged properly the electrocardiogram has a normal appearance The patient was a healthy 23 year old man whose abnormal heart shadow was detected in a mass miniature x ray survey for tuberculosis

the electrocardiogram generally shows right ventricular hypertrophy or persistent incomplete right bundle branch block (Fig 37), but when the degree of stenosis is slight the electrocardiogram may be normal In interatrial septal defect the electrocardiogram generally shows right ventricular hypertrophy or right bundle branch block (Fig 36) but, here again, the tracings may be normal if the defect is small It is a curious fact that atrial septal defects are more commonly associated with intraventricular block (prolonged QRS complex) and auriculoventricular block (prolonged P R interval) than are interventricular septal defects Arrhythmias including auricular fibrillation are not uncommonly seen in interatrial septal defects

In the tetralogy of Fallot there is always an extreme degree of right ventricular hypertrophy and the P waves are apt to be tall and pointed Right ventricular hypertrophy may also be seen in transposition of the great vessels or in anomalous entrance of the pulmonary veins into the right auricle

In patent ductus arteriosus, where the greater mechanical burden is borne by the left ventricle, left ventricular hypertrophy may be detected by the electrocardiogram In many cases where the defect is small, the electrocardiogram may appear normal First degree heart block is not uncommon Coarctation of the aorta and congenital aortic or subaortic stenosis are also apt to be associated with left ventricular hypertrophy (Fig 40) *In interventricular septal* defects the forces are generally balanced so that the electrocardiogram is usually normal, occasionally, however, one ventricle appears to be predominantly overburdened so that either right or left ventricular hypertrophy may be recorded Occasionally the defect appears to intercept the auriculoventricular bundle producing congenital complete heart block, but the latter condition may also occur with an intact septum Congenital complete heart block may be rather well borne, the heart rate is generally more rapid than that seen with acquired complete heart block

CHANGES IN THE FORM OF THE AURICULAR COMPLEX

The auricular complex (P wave) varies in form somewhat in normal individuals In general with faster rates the P wave is larger and with

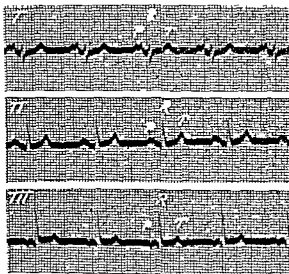


Fig 128 Auricular hypertrophy Note that the P waves in Lead I are broad flat topped and notched The patient had well marked mitral stenosis

slower rates it is smaller This is due to the fact that impulses arise high in the sino-auricular node when the rate is rapid and low in the sino-auricular

node when the rate is slow Generally the P wave shows a much more conspicuous deflection in normal sinus tachycardia than in paroxysmal auricular tachycardia Occasionally this fact may be helpful in making the difficult differential diagnosis between these two types of tachycardia There is one peculiar type of P wave that is present so frequently with mitral stenosis that it is fairly diagnostic It is a wave that is prominent, broad, somewhat flat-topped and notched (Figs 128 and 129) These characteristics are generally displayed in Leads I and II, and often in Lead III the P wave is inverted This type of P wave usually denotes an enlarge-

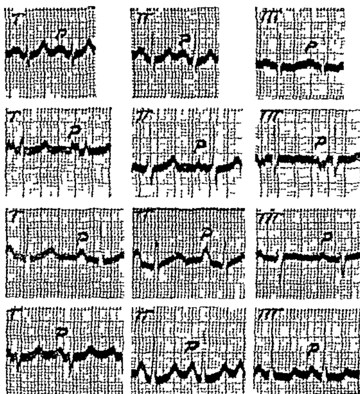


Fig 129 Auricular hypertrophy Four sets of curves showing large broad and notched P waves Note that P waves may be most prominent in Leads I or II and may be inverted in Lead III All four patients had rheumatic mitral stenosis with or without aortic or tricuspid involvement

ment of the auricles, and as this occurs almost exclusively in mitral stenosis it has important clinical value When curves are found, such as are represented in Figures 128 and 129, one can be quite certain that mitral stenosis is present There may be an additional tricuspid stenosis but this never occurs without the presence of mitral stenosis as well Occasionally broad notched P waves of the type described above may be observed during acute myocardial infarction Deviations of the P-R segment have been described in the auricular infarction which may accompany infarction of the ventricle

Sharp, narrow and large P waves may be seen in hyperthyroidism and it is suspected that tricuspid stenosis may have a prominent P wave that is taller and narrower than those just described for mitral stenosis

Often, in association with other evidence of right ventricular hypertrophy, the P waves are tall (2 5 mm or more) and pointed but not broadened in Leads II and III These have been referred to as P pulmonale Whether this change is due to auricular hypertrophy or simply to a peculiar position of the auricles has not been determined This change is frequently seen in pulmonary emphysema

CHANGES IN FORM OF VENTRICULAR COMPLEXES

There now remains for consideration a heterogeneous group of conditions in which the ventricular complex becomes abnormal, and the clinical significance of such changes It will be seen that in some conditions the electrocardiographic findings are most important and helpful, in others they may be only suggestive and frequently the changes may be misleading because they are not sufficiently distinctive

Ventricular Complexes of Low Amplitude

When the height of the R wave or the depth of the S wave is 5 mm or less in all leads it is regarded as abnormal (Figs 130 130*A*) Upward measurements should begin with the top of the isoelectric shadow of the string and those downward from the bottom of it Such low curves may occur with advanced cardiac failure from any cause either valvular or non-valvular with acute coronary occlusion (Fig 130*A*), anemia, myxedema Addison s disease, pericardial effusion or any condition showing considerable edema They are also frequently present even without evidence of heart failure, in patients with well marked emphysema of the lungs The low complexes are either due to the development of an electromotive force of small potential within the heart or to some electrical effect of the edema of tissues around the heart The curves may become larger if the edema disappears or the congestive state improves In myxedema (Fig 131) not only are the QRS waves diminished but the T waves are flat Such curves can return to normal on thyroid therapy When they are found after a coronary occlusion they are apt to persist indefinitely and be compatible with a satisfactory state of health for years In general curves of very low amplitude indicate a fairly grave condition of the heart but there are exceptions to this rule

There are tracings that show ventricular complexes of low amplitude that require a different interpretation They are not apt to be quite so small, although they may be, but they appear otherwise normal (Fig 132) The QRS waves are not notched or spread and the T waves have a normal configuration These points distinguish them from the abnormal ones described above They are found in individuals who are well and show no evidence of organic heart disease Low voltage in the limb leads is more significant if there is associated low voltage in the precordial leads It has been shown that in certain cases with low voltage in the limb leads the

largest deflections occurred in Lead V_2, whereas normally the largest deflections occur in Lead V_4 This suggests not heart disease but a shift of



FIG 130 Abnormal form of ventricular complex (low voltage) Four sets of curves showing QRS complexes of low amplitude in all leads The lowest curves show the normal standardization All four patients had serious heart disease of the coronary artery type

the mean electrical axis of the heart toward the sagittal plane of the body It is evident, therefore, that ventricular complexes of low potential must

be interpreted with due care and should be used only in conjunction with other data as indicative of heart disease

Ventricular Complexes in Coronary Thrombosis and Myocardial Infarction

The most important contribution that electrocardiography has made to medicine is in the diagnosis of coronary thrombosis and myocardial infarc-

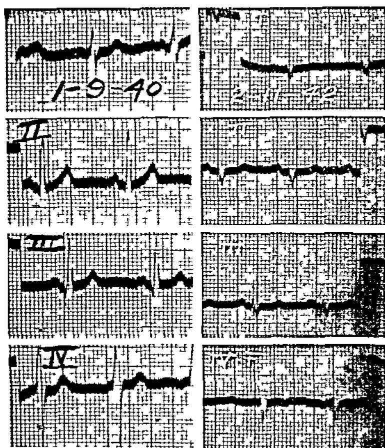


Fig 130*A* Abnormal form of ventricular complex (low voltage) The first set taken January 9 1940 shows nothing very abnormal An attack of acute coronary thrombosis occurred in the summer of 1940 The second set taken February 2 1942 shows marked decrease in amplitude of QRS and T waves The patient had no peripheral edema although there was a moderate right hydrothorax

tion This goes back to the early observations of Pardee when he first called our attention to the displacement of the S-T segment in acute myocardial infarction When the ventricular complexes change after such an attack they do so because of alterations in the musculature supplied by the vessel involved and not because of the thrombosis itself It would be more proper to designate these abnormalities as indicative of myocardial infarction,

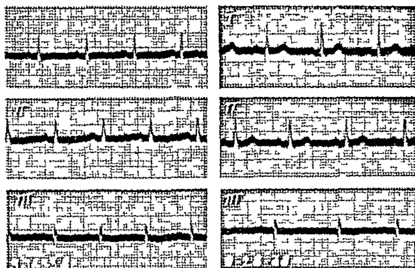


Fig 131 Effect of myxedema The first set was taken September 14, 1938 when the basal metabolic rate was −35 per cent The second set taken fourteen days later, after thyroid therapy, the basal metabolic rate being −10 per cent Note the increase in height of T and R waves

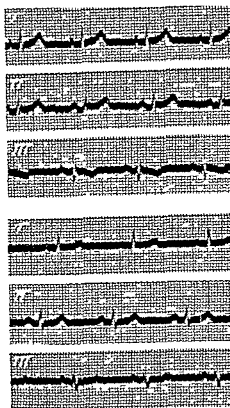


Fig 132 Low voltage Two sets of curves showing QRS waves of low amplitude Note that the general form of the ventricular complexes is normal (upright T_1 and T_2 and sharp R waves) in contrast to those in Figure 130 These two patients had no heart disease

which generally results from coronary thrombosis but may come from disease of the arteries such as narrowing without thrombosis

Myocardial injury produces changes in the electrocardiogram which vary with the degree of injury, the size of the area injured and its location These changes are of two sorts (1) changes in the repolarization process (T wave and RS-T segment), and (2) changes in the depolarization process (QRS complex) We have seen that the T wave is the most labile part of the electrocardiogram, changing readily as the result of chemical, mechanical, anoxic or thermal influences while the QRS complex is relatively fixed and capable of change only by the most severe grades of injury This corresponds to the clinical fact that changes in the T waves of themselves are to be regarded as nonspecific in their implications whereas changes in the QRS complex generally correspond to profound muscle damage

Although recent animal experiments have shown the earliest electrocardiographic consequences of impairment of the blood supply to the heart to be a sharp inversion of the T waves, this is ordinarily not observed clinically since patients are rarely seen at the very onset of an attack of myocardial infarction Two recent cases, personally observed, are of some interest in this connection The first patient clearly had the onset of his acute myocardial infarction while being fluoroscoped An electrocardiogram recorded immediately showed an inverted T_3 with a prolonged Q-T interval Serial electrocardiograms subsequently showed the characteristic sequential changes of acute posterior myocardial infarction Another patient was hospitalized in coma due to a ruptured intracranial aneurysm This patient subsequently showed RS-T segment elevations in the same leads (Fig 133) which originally showed inverted T waves There was no demonstrable infarct at postmortem examination in this case

T wave inversion of this type has been called ischemic in type These are the same sort of T waves that characteristically appear at the margins of an infarct later in the course of an acute episode By and large, however, the earliest changes recorded consist of displacements of the RS-T segments An electrode overlying a subepicardial or transmural lesion records an upward displacement (elevation) of the RS T segment whereas an electrode overlying the opposite healthy ventricular wall records a reciprocal down ward displacement (depression) of the RS-T segment On the other hand, an electrode overlying an acute infarct which is subendocardial in its location records a depressed RS-T segment while an electrode, such as that at Lead aV_R, which faces the ventricular cavity and hence the injured subendocardial layers records an elevated RS-T segment This latter is the same sort of change which may readily be induced transiently in certain individuals with angina pectoris It is difficult to reconcile this clinical phenomenon of easily induced RS T segment depression with the experimental experience that RS-T segment elevations are recorded in animals only on a more prolonged coronary artery ligation than produced the T wave inversion described above It is convenient, however to correlate the three types of electrocardiographic changes with three stages of myocardial impairment, the QRS changes corresponding to myocardial necrosis the

RS-T segment deviations to injury effect and T wave inversion to myocardial ischemia

The RS-T segment displacements occurring during an acute myocardial infarction generally last only a few days, gradually returning to the isoelectric line After a variable interval they are usually accompanied by terminal inversion of the T wave giving rise to a characteristic appearance in the RS-T segment and T wave variously described as a "coronary T wave," 'Pardee T wave, "cove plane T wave," 'round shouldered T wave' or 'upwardly bowed T wave" The Q-T interval in these instances is ordinarily prolonged The T waves may become very deeply inverted and V-shaped, corresponding to this change the opposite ventricular wall may exhibit very tall pointed T waves The T-wave changes generally outlast

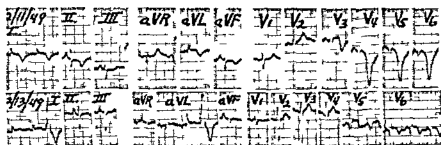


Fig 133 Ischemic T waves RS T and T wave changes without infarction The patient was a 69 year old woman admitted in coma February 9 1949 and died February 16 1949 In the initial set of tracings obtained on February 11 1949 the T waves in Leads I II aV_L, aV_F and V_3 to V_6 have a characteristic 'coronary' contour In Leads V_3 to V_6 the T waves have a waterfall appearance often seen during the early days or weeks of an acute myocardial infarction or in the very earliest stages of experimental coronary artery ligature The tracings obtained on February 13 1949 show elevation of the RS T segments in Leads I aV_L, and V_4 to V_6 with much less pronounced late inversion of the T waves in these same leads Although Q waves were recorded in Leads V_4 to V_6 they are very narrow and very inconspicuous relative to the R waves in the same leads and accordingly are attributable to septal activation and not to myocardial death The patient remained in coma and died two days later Postmortem examination showed a ruptured aneurysm of the circle of Willis but no evidence of myocardial infarction or pericarditis on meticulous microscopic examination

the RS-T segment changes and may persist for weeks, months, or even years but they are apt to lose their characteristic shape and become merely inverted T waves of otherwise normal appearance In time these may become isoelectric or normal upright T waves

Frequently at the same time, but occasionally at variable intervals after these changes in the repolarization process, changes may also appear in the QRS complex These changes probably correspond to actual death of parts of the myocardium which are no longer capable of responding to the activating impulse If the entire thickness of the ventricular wall thus becomes electrically inert, the cavity potentials are transmitted passively through the infarct to an overlying electrode just as they would be through the electrically inactive auriculoventricular valve ring to an electrode (such

as an esophageal electrode at auricular levels) facing the ventricular cavity Since the remaining healthy ventricular myocardium is still activated from endocardium to epicardium, the impulse moves away from the ventricular cavity and a downward deflection is recorded in the cavity The electrode overlying the transmural infarct therefore picks up the same potential and records a QS deflection This QS wave ordinarily has considerable duration, 0 03 second or more from onset to nadir, 0 04 second or more in total breadth If only the inner layers of the myocardium are destroyed and the outer layers are still absolutely or relatively healthy an overlying electrode records first a Q wave, corresponding to unopposed activation of the healthy subendocardial muscle of the opposite wall, followed by an R wave corresponding to activation of the healthy subepicardial muscle under the electrode However, this R wave is not so tall as the original R wave recorded over the same area Therefore the R wave decreases in height and a Q wave appears if the infarct is subendocardial, whereas the R wave disappears to be supplanted by a QS deflection if the infarct is transmural Which of these changes is recorded by an electrode depends upon its relation to the center or margin of the infarct Direct leads record T wave inversions only at the margins of experimental infarcts In clinical myocardial infarction, however, T wave inversion is apt to be recorded in the same ventricular complex in which QS deflections are present, in this case the electrode is a semidirect one and picks up a mixture of the potentials over the infarct and in its vicinity If the area of transmural infarction is small QRS changes may be missed in the twelve lead electrocardiograms now in more general use, or they may be recorded in only one lead Here is another reason for taking multiple leads

The area over which QRS, RS T and T wave changes develop depends, among other considerations, upon the size of the infarct QS or QR waves may be detected over a wide area if the infarct is large Although one might expect that the prognosis is worse in larger than smaller infarcts, we know of no study which establishes this possibility The area showing changes in the ventricular complex depends also upon the orientation of the infarct with relation to the electrodes From the characteristic changes developing in certain leads it is possible to localize an infarct quite accurately This procedure is described at greater length below It must be emphasized however, that at present localization of infarcts is of more academic than practical interest Once decisive evidence of acute myocardial infarction has been obtained, a record of the sequential changes that subsequently occur is of no clinical value, it is a matter of little moment if there is a lateral component to an anterior or posterior myocardial infarct

As an infarct heals the scar is apt to contract to a smaller and smaller area One which produced cavity potentials over perhaps four of the usual precordial points might then become so small that decisive changes are now recorded at only one point If the scar is not located strategically with relation to these precordial points it may not be recorded at all

In contradistinction to the RS-T and T wave changes which may be evanescent and disappear entirely with healing of the infarct, the QRS

changes are relatively fixed Once they appear they are likely to persist, usually for the remainder of the patient's life On rare occasions Q waves appear and disappear This is often difficult to explain By and large the decision regarding *old* myocardial infarction rests upon the detection of characteristic changes in the QRS complexes The electrocardiographic diagnosis of *acute* myocardial infarction, on the other hand, is made on the basis of characteristic sequential changes in the RS-T, T and QRS complexes As indicated below there are some cases of old infarction, especially in association with aneurysm formation in which the QRS, RS-T and T wave changes may remain fixed over months or years Rarely acute myocardial infarction may be associated with characteristic changes in the RS-T segment and T waves only If a good clinical history suggestive of an acute episode is obtained, the diagnosis of acute infarction is justified in patients with these electrocardiographic findings These patients are said generally to do well and on recovery may show no electrocardiographic residue of infarction Some patients go through an entire clinical episode of acute myocardial infarction without developing corroborative electrocardiographic evidence for the diagnosis Others will yield decisive or *suggestive electrocardiographic findings only after several tracings have* been recorded In one patient electrocardiographic changes did not appear until the third week of a typical clinical episode Hence, although, as stated above, serial tracings are not helpful once the diagnosis has been established, they are imperative until that diagnosis has been made or another diagnosis has been substituted

Anterolateral Infarction (Antero-apical Infarction) When an infarct involves both the anterior and lateral aspects of the heart changes of the type described above are generally recorded between Leads V_3 and V_6 A clue to anterolateral infarction is a decrease in the height of the R wave as the electrode is moved to the left of Leads V_1 or V_2, but this is not conclusive QS deflections corresponding to transmural infarction may be present over one, two or three positions At one or more positions to the left of the QS wave and corresponding to a subendocardial extension of the infarct QR waves may be recorded (Figs 134 to 136) RS-T and T waves changes of the type described above generally occur in association with the QRS changes The exact leads in which these changes are recorded vary with the position of the heart and of the infarct with relation to the precordial leads and with the precision with which the electrodes are placed at the same precordial positions at each recording of the electrocardiogram

In these cases the infarct generally extends far enough laterally to be registered at the left shoulder especially if the heart lies in a horizontal electrical position Even if the heart is not horizontal the infarct may extend high enough on the lateral surface to be recorded at the left shoulder Accordingly changes in the ventricular complex are apt to be recorded in Lead aV_L as well as in the lateral precordial leads Since the potentials of Lead aV_L are written with unchanged polarity in Lead I, this lead shows prominent Q waves, elevated RS-T segments and characteristic inversion

of the T waves On the other hand since Lead aV_L has a reversed polarity in its contribution to Lead III, Lead III is apt to develop smaller R waves, deeper S waves, depressed RS-T segments and upright T waves Thus

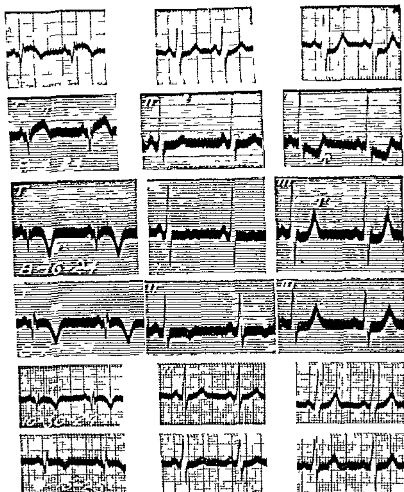


Fig 134 Anterolateral myocardial infarction A series of tracings following an attack of acute coronary thrombosis which occurred July 20 1924 Note that the high take off of the T wave and the subsequent sharp inversion occur in Lead I Somewhat similar changes but in the opposite direction occur in T_3 Also there is a distinct Q_1 which persists even the following year These tracings were recorded before the advent of chest leads but probably would be associated with changes in Lead aV_L and in the left lateral chest leads similar to those recorded in Lead I and therefore probably represent infarction of the anterolateral portion of the left ventricle (Author s article in Oxford Loose Leaf Medicine vol II)

Leads I and III may have a mirror image relationship as a result simply of the particular galvanometer poles through which left shoulder potentials are routed Another cause of reciprocal changes in the electrocardiogram is described below under posterior myocardial infarction

If the infarct is so oriented that only T wave changes are transmitted to the left shoulder, Lead I will record only an inverted T wave This T wave might or might not have a so-called 'coronary' contour Thus only dubious

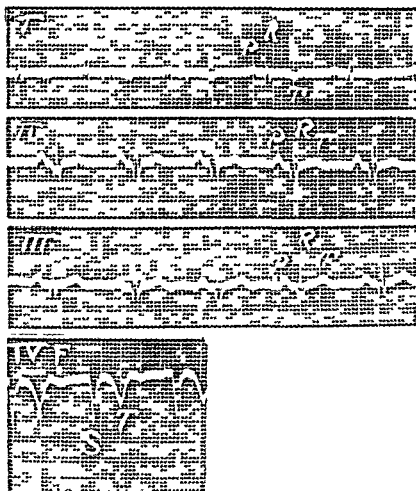


Fig 135 Anterolateral myocardial infarction Note the rounded and slightly inverted T_1 and a small Q_1 Lead IV_F shows deep QS (no R) wave and a deeply inverted T wave (Normally there should be an R and upright T wave in this lead) The attack was considered as due to gastritis three weeks before these tracings were made, because of vomiting for twenty four hours and distress over the ensiform These tracings were taken when a single precordial lead was routine The existence of a Q_1 indicates that the subendocardial extension of the infarct extends far enough laterally to be recorded in Lead aV_L The existence of a QS_{IV_F} indicates that the transmural part of the infarct is located near the cardiac apex

evidence for infarction will be present in the standard leads when decisive evidence is recorded in the precordial leads

Following healing of the infarct the only residuals may be a prominent Q_I and the presence of decreasing R waves, of absent R waves where R waves are ordinarily recorded, or of QS or QR complexes in the lateral

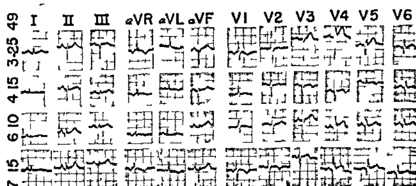


Fig 136 Acute anterolateral changes possibly superimposed upon old postero lateral myocardial infarct (?) The patient was a 62 year old widow without a previous history of angina pectoris or myocardial infarction who developed severe crushing midchest pain five days before admission The initial set of tracings recorded shortly after admission showed prominent QS deflections in Leads III and aV_F and broad prominent Q waves in Leads II and V_4 to V_6 The RS T segments were elevated in Leads V_4 and V_5 and slightly in Leads III and aV_F These changes indicate myocardial infarction probably acute During the first week the patient developed an elevated sedimentation rate and leukocytosis The second set of tracings taken three weeks later show persistence of the QRS changes return of the displaced RS T segments toward the isoelectric line and a terminal dip in the T waves in Leads I and V_2 to V_6 The next two sets of tracings taken two and three months later show return to the appearance noted in the original tracings The patient recovered In this case the RS T and T wave changes were most marked anterolaterally while the QRS changes persisted posterolater ally It is possible that QRS changes were present posterolaterally before the present attack and that the only recent electrocardiographic developments were ischemic anterolateral changes The persistent changes four months after the attack suggest ventricular aneurysm This series illustrates the occasional difficulty in interpreting tracings where more than one infarct of varying duration may be involved



Fig 137 Acute anteroseptal infarction The upper set shows the three standard leads that are not definitely diagnostic of myocardial infarction The lower set of six bipolar chest leads shows absence of an initial upward deflection in CF_{1-5} and an elevation of RS T in CF_1 to CF_4 with late inversion of T waves These are diagnostic of anteroseptal infarction The patient was a man 75 years old with a known old gastric ulcer who developed pain in the xiphoid region which was first misinterpreted as due to a subacute perforation of the stomach

precordium Generally these changes are much more readily appreciated in the precordial than in the standard or unipolar limb leads

Anteroseptal Infarction Here the changes are recorded in the region of the sternum, usually in Lead V_1 through V_4 (Figs 137 to 141) Changes in Lead V_1 and V_2 are said to be more commonly associated with infarction of the septum whereas lesions located nearer the apex are likely to be asso

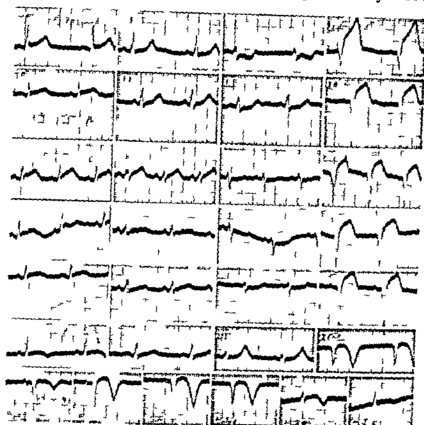


Fig 138 Acute anteroseptal myocardial infarction This series of tracings shows three standard leads and CF_4 beginning about four hours after the onset The standard leads go through the customary changes of anterior infarction i e elevation of RS T_1 and a depressed RS T_3 becoming a sharply upright T_3 Note that the first set shows a very high take off of RS T_{IV_F} which remains elevated for over a week and finally becomes sharply inverted The R_{IV_F} is absent through out This is characteristic of anteroseptal myocardial infarction The lowest set shows the six precordial leads CF_1 to CF_6 Note that R is absent and T is sharply inverted over the first four positions indicating a large area of infarction The patient was a man 41 years old who recovered and became symptomless

ciated with a small R wave in Lead V_1, giving way to QS or QR complexes in Leads V_2 and V_3 A decrease in the size of the R wave as the electrode moves to the left is presumptive but not conclusive evidence of old anteroseptal infarct (Fig 141) In rare cases elevation of the RS-T segments and inverted T waves may be inscribed at Leads V_1 and V_2 in the absence of more distinctive QRS changes than are ordinarily present in these areas

Fortunately myocardial infarction is not prone to develop in the age and sex groups which may normally show inverted T waves over the right ventricle

RS T and T wave changes may be recorded to the left of Leads V3 or V4 but only rarely are diagnostic changes reflected in Lead aVL or Lead I The reason for this is that, by and large, the electrical forces are oriented in an anteroposterior plane and have little or no projection upon the frontal

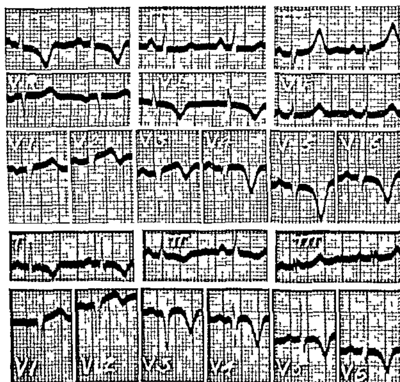


FIG 139 Acute anteroseptal myocardial infarction The upper three strips taken October 5 1941 show the three standard leads the three unipolar limb leads (V_R V_L and V_F) and the six precordial leads (V_1 to V_6) Note the absence of R waves in V_1 to V_3 and the gradual appearance of dipping and inversion of T from V_2 to V_6 The lower two strips were made October 27 1941 and show slight regression of the abnormalities The patient was a woman 53 years of age who had a coronary thrombosis two months before and another twelve hours before the first tracing was made Recovery was satisfactory

plane of the body Infarcts in this location were particularly liable to be missed when only the three conventional leads were in use They can also be missed if one takes only a single precordial lead located to the left of the infarct

Occasionally it is difficult to decide from leads taken over the right precordium whether old anteroseptal infarction is present Since the R wave may normally be absent or small in Leads V_1 and V_2, and since R waves

may be small or absent over a larger area (V_1 through V_3 or even V_4) in individuals with left ventricular hypertrophy or complete or incomplete left bundle branch block it may be difficult or impossible to decide whether an old anteroseptal infarct is present In such cases it may be necessary to

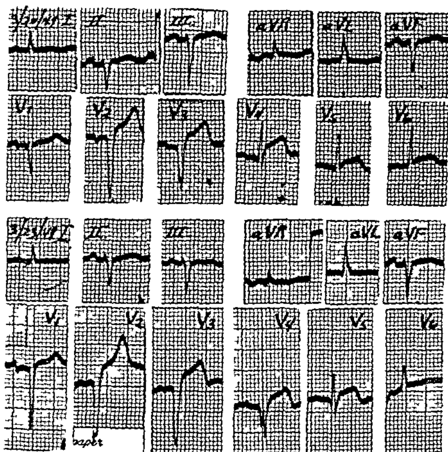


Fig 140 Acute anteroseptal myocardial infarct The initial set of tracings shows the characteristic changes of anteroseptal infarction interpreted as of uncertain duration. Late inversion of the T waves in Leads V_3 to V_5 of the second set of tracings suggested that the process was acute and the QR complex in Lead V_5 indicated subendocardial extension of the infarct The patient was a 78 year old man who had a typical clinical attack of coronary thrombosis following a fracture of the wrist He died five days after the second set of tracings was recorded Postmortem examination showed a large anteroseptal infarct extending almost the entire distance from the apex to the auriculoventricular sulcus with subendocardial hemorrhage and involving the anterior half of the septum Popliteal thrombophlebitis and a large pulmonary embolus to the right lower lobe of the lung were also found

report that all of the changes can be accounted for by left ventricular hyper trophy, for example, but that old anteroseptal infarct cannot be excluded

High Lateral Infarction Localization of an infarct to the upper left border of the heart, sparing the apex and not detectable with certainty in the usual six precordial leads is rare In cases of this sort a clue to the

existence of high lateral infarction may be the presence of a prominent Q wave followed by an R wave at the left shoulder Some difficulty is encountered in evaluating this finding in vertical hearts which may also show a prominent Q or QS wave at Lead aVL The finding of an inverted P wave preceding this QRS complex would be more consistent with a vertically placed heart, for then auricular as well as ventricular depolarization proceeds away from the left shoulder One would then have to conclude that the left shoulder electrode must face the auriculoventricular valve ring through its auricular aspect, and that the Q wave inscribed at the left shoulder lead represents cavity potential transmitted through the valve ring If, on the other hand, the P wave is upright, auricular depolar-

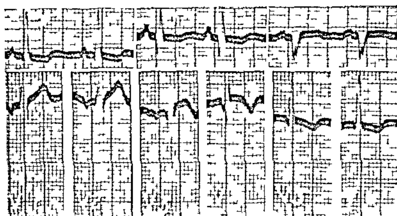


FIG 141 Old anteroseptal myocardial infarction The upper set shows the three standard leads and the lower the six precordial leads taken February 16 1943 Note that the R wave gradually decreases in size from V_1 to V_3 so that it is absent at V_3 There are also changes in T in V_4 to V_6 The broad prominent Q wave in Lead II and the notched QS wave in Lead III may be explained by a coexistent old posterior lesion by anteroposterior (Roesler Dressler) infarction or if the heart were in an extreme vertical position by transmission of the cavity potentials from the anterior surface of the heart to the left leg The patient was a man 39 years old with severe chronic arthritis who had an acute coronary thrombosis in April of 1942

ization must proceed toward the electrode Unless the electrode happens to lie in precise relation to the epicardial aspect of the auriculoventricular sulcus, one would be justified in assuming that it overlay the infarcted free wall of the left ventricle, passively transmitting cavity potentials to it Generally a final decision as to high lateral infarction cannot be made from this finding However, if additional leads are then taken in the second third and fourth interspaces in the midclavicular in the anterior and midaxillary lines, one may find unequivocal evidence of infarction in broad prominent Q waves with or without RS-T and T wave changes, distributed over a considerable area In our experience high lateral infarcts have constituted portions of infarcts already easily detected elsewhere but they

have been reported in the literature as lesions localized to the left upper margin of the heart

Posterior Myocardial Infarction The posterior aspect of the heart lies upon the cupola formed by the left leaf of the diaphragm The potentials of the posterior wall are transmitted through the diaphragm to the left leg, which is relatively remote from the heart Lead aV_F therefore records the composite potentials of the posterior, and to a lesser extent, of other aspects of the heart Hence posterior wall infarcts are generally detected in Lead aV_F Lead aV_F contributes in a positive way to the potentials of both

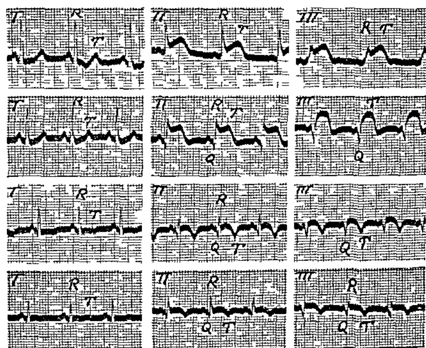


Fig 142 Acute posterior myocardial infarct Attack June 14 1934 four hours before first set of curves Subsequent tracings taken June 20 1934 July 6 1934 and August 17 1934 Note very high take off with monophasic action current in Lead III of first set Note also that Q waves have not yet developed in the first set The RS T segment returns subsequently to the isoelectric line and the T wave inverts A Q_2 and Q_3 gradually develop and persist

Leads II and III Other things being equal, these two standard leads may also detect the changes of posterior wall infarction (Figs 142 to 145) Until the advent of unipolar lead electrocardiography prominent Q waves, ele vated RS-T segments and inverted T waves in Lead III, and to a lesser extent, in Lead II as well have been the evidence upon which the diagnosis has been made However, Leads II and III are composite leads A Q wave in Lead III may result from a Q_{aV_F} or from an R_{aV_L} The use of these unipolar limb leads enables one to decide, at a glance, which of these two possibilities explains Q_3 and therefore, *in large measure*, enables one to decide whether a prominent Q_3 is the result of posterior myocardial infarc-

tion or of left ventricular epicardial potentials transmitted to the left shoulder It is in this differentiation that the unipolar limb leads have their greatest field of usefulness In the decision of insurability, for example a prominent Q3 may be the only residual of a previous posterior infarct If, with such a finding a prominent R_{aV_L} but no Q_{aV_F} (Fig 27) or an inconspicuous Q_{aV_F} are found, one may dismiss the Q3 as evidence of an old posterior scar In the same circumstances a broad, prominent Q_{aV_F} would

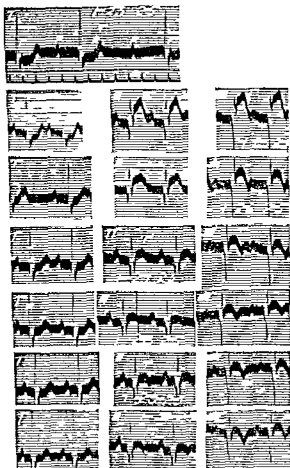


Fig 143 *A* Legend on page 470

be decisive evidence of old posterior wall infarction (Fig 38) Authorities differ as to just what yardstick should be used to decide whether Q_{aV_F} is significant We have adopted the criteria of Myers who states that if QRS has a total deflection of 5 mm or more and the Q wave measures 25 per cent or more of the R wave in that lead and the Q wave measures 0 03 second or more from onset to nadir, the unequivocal diagnosis of posterior wall infarction can be made If only one of these two criteria is present the tracing must be regarded as borderline and consistent with that diagnosis

In our experience, if the Q wave measures 0 04 second from onset to nadir, posterior infarction is probable It is dangerous to draw conclusions from the form of the QRS complex if the total excursion is less than 5 mm In

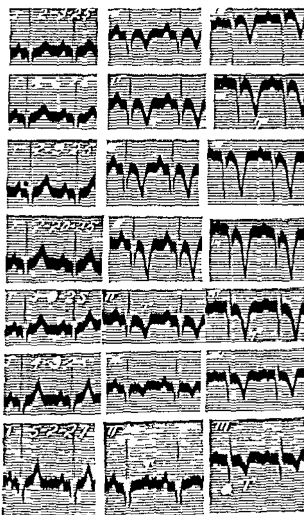


Fig 143 *A* and *B* Acute posterior myocardial infarction Attack of coronary thrombosis on January 12 1925 Note the sequence of changes in the ventricular complexes beginning with a high take-off of the RS T segment in Lead III gradually becoming lower and dipped and finally marked inversion of T_3 Partial heart block was present in the early days A prominent Q_2 and Q_3 developed and persisted If unipolar limb leads were taken in this case Lead aV_F would certainly have recorded the same type of changes as those recorded in Leads II and III The patient did well for five years after the attack (Author s article in Oxford Loose Leaf Medicine vol II)

that case one may well be dealing with the ' transitional zone, recording the composite potentials of both the right and left ventricle In any case the associated presence of RS-T and T wave changes enhances the value of changes in the QRS complex which of themselves may be of dubiou

significance The important point should be stressed that even with the help offered by Lead aV_F it still may be necessary to report equivocal findings The vast majority of cases showing prominent Q_3 but no significant Q_{aV_F} do not have posterior infarction, but we have observed a few excep-

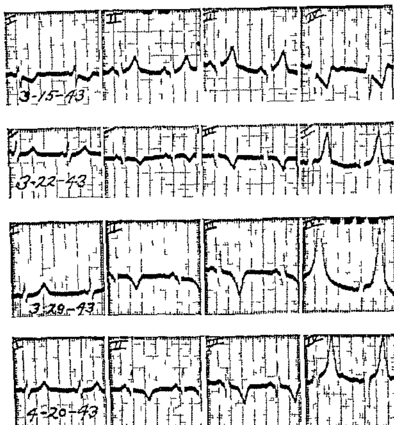


Fig 144 Acute posterior myocardial infarction The first set of tracings was taken six hours after the onset of a typical attack of coronary thrombosis Note depressed RS T segment in Leads I and IV_F with elevation of RS T segment in Leads II and III a distinct Q and Q_3 and then the gradual development of sharp inversion of T_2 and T_3 with a marked exaggeration of the upright T wave in Lead IV_F These tracings illustrate the two respects in which the precordial leads may be of possible help in the diagnosis of posterior infarction namely depression of the RS T segment and the development of very tall T waves in one or more precordial leads These changes may be the earliest clue to that diagnosis The patient was a man 42 years old who while shoveling sand had severe pain in the chest radiating down both arms He had slight fever leukocytosis increased sedimentation rate and the blood cholesterol was 410 Recovery was excellent

tional cases in which posterior infarction was present under these circumstances In these cases, however, the development of associated RS-T changes generally was sufficient to establish the diagnosis It is possible that in these cases the patient died before the QRS changes had sufficient time to develop, that the potentials of the infarct were directed at right

angles to the frontal plane of the body, or that the infarct was not oriented toward the left leg because of some other peculiarity in the electrical position of the heart A prominent QS complex in Lead aV_F is much more difficult to evaluate This appearance is occasionally seen with left ventricular hypertrophy in the absence of posterior infarction It has been claimed that this is due to the transmission of cavity potentials to the left

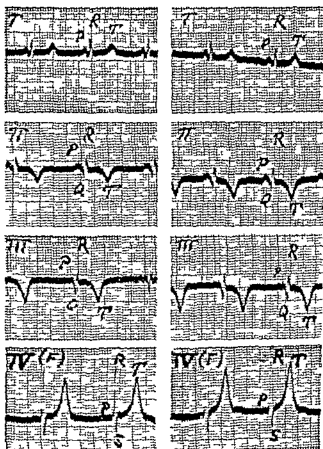


Fig 145 Acute posterior myocardial infarction The attack occurred June 24 1934 The first tracing was made July 11 1934 the second July 18 1934 Note rounded and dipped T_2 and T_3 Lead IV_F shows a normal R wave and an exaggerated upright T wave Similar T waves may be observed in potassium intoxication but the T waves in the limb leads are apt to be pointed and upright in that condition rather than inverted as here

leg but just how this can come about is not clear The point, however, is that a prominent Q_{aV_F} is likely to signify posterior infarction only if it is followed by an R wave, however small

The precordial leads may occasionally be of some help during the acute stage of posterior myocardial infarction Two types of change, singly or in combination, may be present These consist of the presence of tall T waves or depressed RS-T segments (Figs 144 and 145) The former are to be dis-

tinguished from the tall, narrower, pointed T waves which occur in potassium poisoning and the latter from the depressed RS-T segments which can develop in subendocardial ischemia or infarction involving the anterior wall of the heart The described changes in the precordial leads in posterior infarction may actually precede the development of QRS, RS-T or T waves changes in Leads aV$_F$, II or III They are apt to be very transitory in their appearance Hence the precordial leads are not of much help in the diagnosis of *old* posterior myocardial infarction

In the section on anterolateral infarction it was pointed out that reciprocal changes may develop in Leads I and III merely as a consequence of the fact that left shoulder potentials are routed through the electrocardiograph in one direction in writing one of these leads and in the opposite direction in writing the other However, reciprocal changes in the electrocardiogram can also develop because one electrode may face one aspect of a current of injury while another electrode faces its opposite aspect This is the explanation for the reciprocal changes described in the preceding paragraph In certain cases of acute posterior myocardial infarction the left leg electrode faces the epicardial aspect of the infarct and records an elevation of the RS-T segment, whereas one or more of the precordial leads may face the endocardial aspect of the infarct and therefore records depressed RS-T segments

It should be obvious that because of the distance of the left leg or, for that matter, of the groin from the heart, one lacks in Lead aV$_F$ the ability to delineate precisely the presence and extent of a posterior infarct that the multiple precordial leads possess in the detection of anterior infarcts With the usual precordial leads tapping the potentials approximately over a horizontal plane on the chest one can demarcate anteroseptal, anterolateral, massive anterior or, with additional leads, high lateral lesions No such precision is possible with posterior wall infarcts Esophageal leads recorded at ventricular levels indicating changes in a more vertical plane in the body, may offer some help in this regard but the procedure is somewhat annoying and should not be tried if the patient's condition is too critical In chronic cases in which Lead aV$_F$ gives equivocal evidence of posterior infarction our experience has shown that esophageal leads are apt to leave one just about as much in doubt This deserves further study Mere repetition of the entire set of tracings which may happen to change the relationship of the heart to the left leg, is less disturbing and, in many cases, reveals decisive changes one way or the other The practice of Goldberger of having the patient take a deep breath during the recording of Lead aV$_F$ may accomplish the same purpose Generally speaking, esophageal leads are much more helpful in the study of electrical activity in the auricles

Normally, as the electrode rises from the lower esophagus, near the diaphragm, the auricular waves are very slight until the electrode reaches a point just behind the left auricle Then very sharp P waves appear (Fig 146) This makes it very simple to know that the electrode is lower than the auricle and therefore adjacent to the posterior portion of the left ven-

tricle From this latter position the ventricular complex normally should display a prominent R wave, possibly preceded by a very small Q and followed by an upright T If the posterior wall is infarcted the ventricular complex will show the same changes that are found with an anterior infarction when the precordial electrode is placed over the precordium, i e, an absent R, an inverted T (Fig 146), and possibly a deviation of the S-T segment during the acute stages



Fig 146 Esophageal leads—posterior infarction The upper set shows three standard leads suggestive of a posterior infarction The next set shows the six precordial leads using the central terminal and gives no positive evidence of infarction The third set was taken *with an electrode in the esophagus at various levels* from the external nasal orifice (number of centimeters) Note that at distance E 71 to E 50, when the electrode was adjacent to the left ventricle there were no R waves and T waves were inverted Contrast these with esophageal curves from a normal individual shown in the lowest set (E 56 and E 52) The appearance of sharp P waves marks the presence of the electrode behind the left auricle and only ventricular complexes below this point are significant The upper three tracings afford positive proof of posterior infarction

The various grades of auriculoventricular heart block may occur with anterior myocardial infarction but, because the bundle of His and associated tissues are irrigated through the same artery that generally nourishes the posterior aspect of the heart, namely the right coronary artery, this complication is much more frequent with posterior infarction

Posterolateral Infarction If the posterior as well as the lateral aspects of the heart are infarcted the potential changes produced by the infarct are reflected to Lead V_6 or Leads V_5 and V_6 as well as to Lead aV_F Figures 147–149 are examples of infarcts of this type Since these leads may be located about 180 degrees around the ventricles from Leads V_1 and V_2,

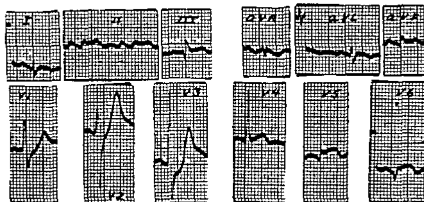


Fig 147 Acute posterolateral myocardial infarction The tracings show prominent Q waves elevated RS T segments and terminal inversion of T waves in Leads II III aV_F V_5 and V_6 Note reciprocal depression of the RS T segments in Leads $V_{1\,3}$ The patient was a 62 year old diabetic who had a typical coronary occlusion two days previously He died on the morning of admission Postmortem examination showed a large posterolateral infarct and a thrombus in the circumflex branch of the left coronary artery which in this case supplied the posterior wall of the heart

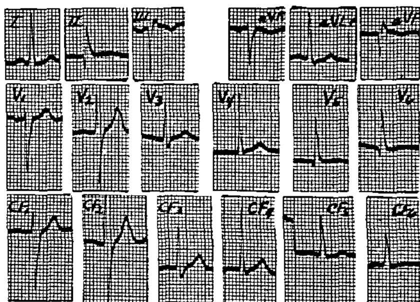


Fig 148 Old posterolateral myocardial infarction The broad prominent Q waves in Leads II and III are associated with a broad prominent Q_{aV_F} This establishes the existence of a posterior infarct Intraventricular block is also present (QRS = 0 11 second) Note how the Q waves present in Leads V_5 and V_6 are missed in Leads CF_5 and CF_6 This results from the large Q wave in Lead aV_F which contributes an R wave to the bipolar CF leads The patient was a 60 year old man who had had an acute coronary occlusion one year previously and died of a severe viral pneumonia Postmortem examination showed a large posterolateral scar In this case the failure to detect the lateral component of the infarct by the CF leads was not important clinically

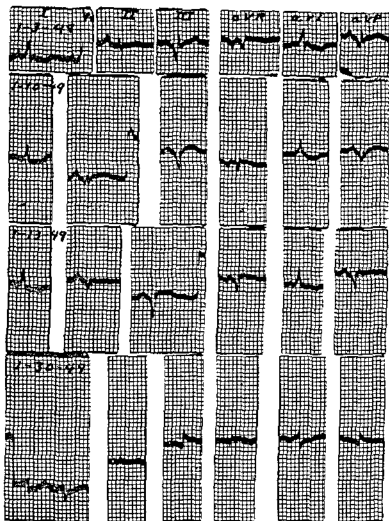


Fig 149 See legend on opposite page

one may find reciprocal changes in the latter leads (Fig 147) Thus the deep Q waves and elevated RS-T segments of Leads aV_F, V_5 and V_6 may be associated with tall R waves and depressed RS-T segments in Leads V_1 and V_2 In Figure 149 the development of this tall R wave over the right side of the precordium, resulting from unbalanced forces (the failure of the posterior wall to be normally activated accounting for the release of these R waves) obscured the previous evidence of old anteroseptal infarction This phenomenon probably accounts for many of the failures to detect an old infarct in the face of fresh infarction or to detect multiple old infarcts

It is important to bear in mind that, in addition to the possibility of posterolateral infarction, changes suggesting coincident posterior and anterior infarction may be produced in two other ways (1) In the so called anteroposterior (Roesler-Dressler) infarct, which involves the interventricular septum and the contiguous portions of the posterior and anterior

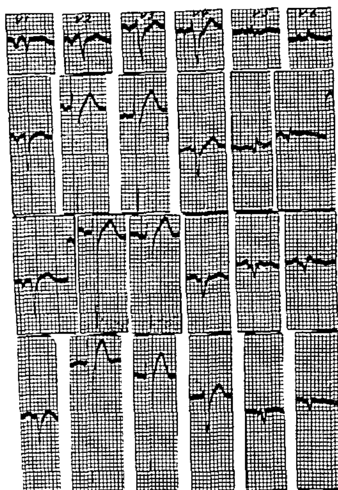


FIG 149 (Continued) Acute posterolateral infarction masking old anteroseptal infarction The patient was a 66 year old man with long standing angina pectoris and a severe episode of prolonged chest pain seven years previously who was admitted for recurrent chest pain The initial set of tracings showed a broad prominent Q wave in Leads II III and aV_F regarded as evidence of posterior myocardial infarction of uncertain duration The R wave was absent in Lead V_1 and small in Leads V_2 and V_3 suggesting old anteroseptal infarct Subsequent tracings showed R waves of considerable magnitude in Leads V_2 and V_3 Elevation of the RS T segments in Leads III aV_F and V_5 and V_6 suggested fresh posterolateral infarction Postmortem examination showed a large acute infarct of the lateral wall of the heart midway between the apex and the auriculoventricular sulcus and an old anteroseptal scar In this case the initial set of curves showed uncertain evidence and the subsequent curves no evidence of old anterior scarring The mechanism of balance of forces is one way in which multiple infarcts may be missed

ventricular walls, deep Q waves elevated RS T segments and T waves may be recorded in Leads aV_F, II, III, and in general one or more of the leads between Lead V_1 and Lead V_4 (2) If the heart is in an extreme ver-

tical position the potentials of the antero-apical portion of the heart may theoretically be transmitted through the diaphragm to the left leg and accordingly to Leads aV_F, II and III Similar unusual rotations may account for variations in the standard descriptions of the electrocardiographic changes described here It must be pointed out, however, that it is frequently quite difficult to define the electrical position of infarcted hearts Infarction may render a considerable part of the ventricles electrically inactive and thus give preponderant electrical weight to the uninfarcted part of the heart, quite independently of the position of the heart The estimation of the electrical position of the heart depends upon the definition of right and left ventricular potentials and their reflection to the extremities In infarction, changes from one type of potential to another may be due to a transition not from one ventricle to another but from an infarcted to an uninfarcted area, and in any given case it may be impossible to decide which With this limitation in mind it is frequently possible to estimate the electrical position of the infarcted heart

A word of warning about the accuracy of the electrocardiographic diagnosis of myocardial infarction, a warning perhaps not adequately sounded in recent publications While it is true that the introduction of multiple chest leads and unipolar limb and chest leads has increased the accuracy of detection and localization of infarcts, its more important contribution has been in a better understanding of the electrophysical principles involved As before the advent of these more elaborate studies, infarction, especially old myocardial scars, can still be missed by the electrocardiogram Thus, in a recent electrocardiographic-pathologic correlation, although the electrocardiographic diagnosis of acute anterior or posterior myocardial infarction was invariably substantiated at postmortem examination, a small minority of cases with an acute anatomic lesion failed to show diagnostic electrocardiographic evidence for fresh infarction In those few cases in which the diagnosis was missed the electrocardiogram was abnormal but nonspecific or the changes of acute infarction were obscured by bundle branch block, left ventricular hypertrophy or acute infarction elsewhere in the heart In the cases of healed myocardial infarction, moreover, although the electrocardiographic diagnosis was almost invariably substantiated when made (the single exception showed decreasing R waves in Leads V_1 to V_3 with no scar at autopsy), the greater majority of scars found on the postmortem table were missed electrocardiographically Here again the electrocardiogram was abnormal but not diagnostic and the changes were obscured, among other causes, by left ventricular hypertrophy, bundle branch block or old or recent infarction elsewhere in the heart

Myocardial Infarction with Bundle Branch Block Pure infarction of the right ventricle is almost unknown When part of the wall of the right ventricle is infarcted anteriorly or posteriorly this is but a small component of an infarct involving largely the contiguous interventricular septum and left ventricle By and large, then, myocardial infarction is a left ventricular affair To understand the electrocardiographic changes which occur in

myocardial infarction complicated by or complicating bundle branch block it is important to bear this point in mind

In right bundle branch block the septum is activated, as normally, from its left to its right side Therefore the left ventricular cavity potential shows an initial downward deflection, if now the free wall of the left ventricle is infarcted this downward deflection (Q wave) may be transmitted through a transmural infarct to Lead aVF if the infarct happens to be posterior in location or to the left side of the precordium if the infarct happens to be anterior in location (Figs 150 to 153) Hence the QRS changes of left ventricular infarction persist with right bundle branch block Furthermore, since the bundle branch of the ventricle (right) opposite that in which we are interested is the one which is blocked there is nothing to mask the

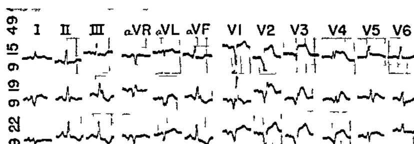


Fig 150 Transient right bundle branch block complicating acute anteroseptal myocardial infarction The patient was a 54 year old robust fireman without previous angina pectoris who was hospitalized because of severe chest pain and shock Tracings on admission showed elevated RS T segments in Leads V_1 to V_5 Note monophasic' action currents in Leads V_2 to V_4 This extreme degree of current of injury is no more ominous than slight but definite RS T segment elevations In the second set of tracings the original evidence of acute anteroseptal infarction i e deep Q waves and elevated RS T segments is still present despite the supervention of right bundle branch block Three days later the right bundle branch block disappeared The patient died on his eleventh hospital day Postmortem examination showed a massive anteroseptal infarct principally involving the septum There was also an aneurysm of the apical part of the left ventricle

development of the other electrocardiographic features of left ventricular infarction, namely RS-T segment and T wave changes Hence the changes of myocardial infarction may be expected to remain in spite of the persistence or inception of right bundle branch block

When on the other hand left bundle branch block is present, the electrocardiogram generally fails to show the evidence of left ventricular infarction In left bundle branch block septal activation proceeds from its right to its left side Therefore the left ventricular cavity records an initial upward (R) deflection which is transmitted through a transmural infarct to the left precordium or the left leg, depending upon the location of the infarct The loss of a deep broad cavity Q wave with the development of left bundle branch block is illustrated in Figure 35 If this patient had been first seen at the time of the second set of tracings the diagnosis of posterior myo-

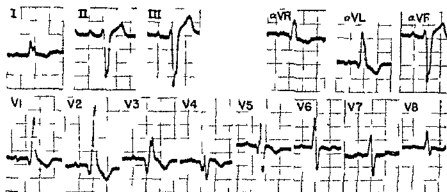


Fig 151 Right bundle branch block with old anteroseptal myocardial infarction QRS duration 0 14 second Broad Q waves and late intrinsicoid deflections are recorded over the right ventricle The Q waves can be explained by septal infarction permitting the passive transmission of left ventricular potentials to the right side of the precordium As in Figure 34 the upright QRS and inverted T wave in Lead I might mislead one into the diagnosis of left bundle branch block but the late intrinsicoid deflections over the right side of the precordium establish the diagnosis of right bundle branch block The probable explanation for this is the transmission of right ventricular potentials to the left shoulder Note how left ventricular potentials are not recorded until Lead V_8 is reached The patient was a 71 year old man with angina pectoris for twenty two years and congestive heart failure since a definite episode of myocardial infarction seven years before these tracings were taken

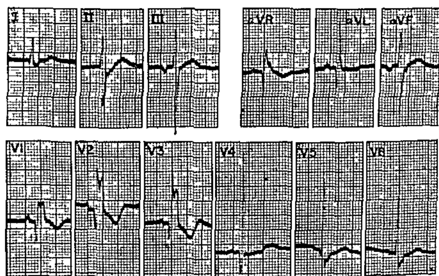


Fig 152 Right bundle branch block with old anteroseptal myocardial infarction The duration of the QRS complex is 0 12 second Late intrinsicoid deflections are recorded over the right ventricle The Q waves are passively transmitted across the septum from the left ventricle to the right side of the precordium The patient was an 82 year old man with a chronic duodenal ulcer He gave no history suggesting angina pectoris or coronary occlusion but he showed evidence of cerebral softening and therefore was an unreliable informant Here the electrocardiogram gave decisive evidence not otherwise obtained

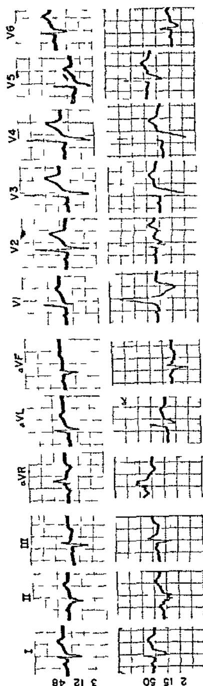


Fig 153 Old posterior myocardial infarction complicated by right bundle branch block The initial set of tracings (3/12/48) shows conclusive evidence of *old posterior myocardial infarction (broad prominent* Q_2 Q_3 and Q_{V_F}) The QRS duration is 0 10 second There is right axis deviation but the prominent R_{V_1} is not an evidence of right ventricular hypertrophy it indicates rather unusual rotation of the transitional zone to the right The septum probably lies to the right of V_1 The position of the heart is indeterminate but the apex is apparently displaced posteriorly as indicated by the tall R_{V_R} and the deep S waves over the entire precordium In the second set of curves (2/15/50) the evidence of old posterior myocardial infarction persists despite the development of right bundle branch block The position of the heart is unchanged Note how the initial 0 05 or 0 06 second of ventricular activation is the same whether or not right bundle branch block is present Note the prolongation of the Q T interval in the second set of curves This results from staggered activation of the ventricles and not from an inherent change in the ventricular muscle

cardial infarction could not have been made on electrocardiographic grounds Since moreover, the left ventricle is the delayed ventricle the slurred turret top portion of the ventricular complex generally encroaches

upon the area in which RS-T segment elevations may be expected to develop The RS-T segment depression and T wave inversion which are present over the blocked left ventricle may be explained as changes "secondary' to the bundle branch block and to the abnormal direction of activation

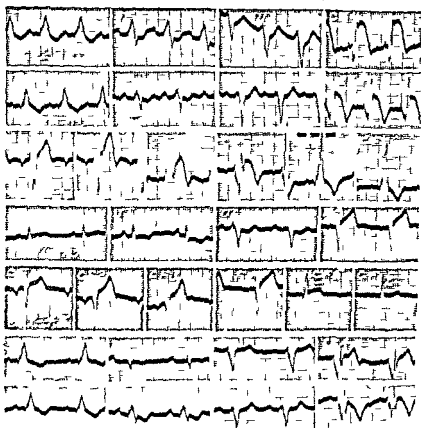


Fig 154 Anterior myocardial infarction with transient left bundle branch block The first set was made a few hours after the onset of an attack of acute coronary thrombosis Note the left bundle branch block Lead CF_4 is most unusual in that it shows a very high RS T junction This is extremely rare in left bundle branch block and is very suspicious of acute anteroseptal infarction The six precordial leads on June 4 1943 show a late intrinsicoid deflection (arrow) over the left ventricle confirming the diagnosis of left bundle branch block Curves on June 7 1943 show that the left bundle branch block is gone Now the intrinsicoid deflection (arrow) is early The lowest set shows that finally T_{CF_4} becomes sharply inverted The patient was a man 75 years of age who had an operation for hernia under spinal anesthesia Preoperative blood pressure was 190 systolic and 110 diastolic, but quickly became imperceptible One hour after the fall in blood pressure a operation was completed pain in the chest was first felt The patient then ran a typical course of acute coronary thrombosis and recovered satisfactorily

of the heart Hence one does not generally get much help from the RS-T segment or T wave changes in left bundle branch block If, on the other hand, as rarely happens, the opposite type of RS-T segment deviation, namely elevation, is observed over the blocked left ventricle, one may

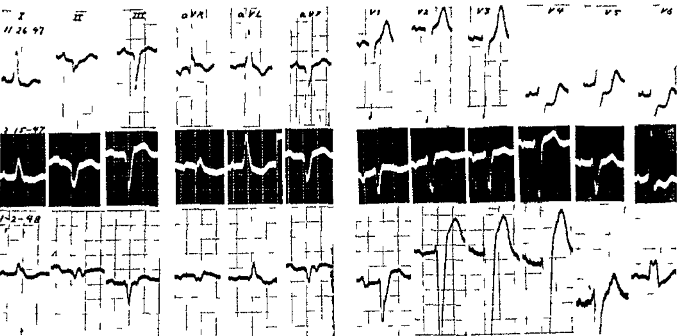


Fig 155 Acute posterior infarction complicated by left bundle branch block and pulmonary infarction A 65 year old man with long standing angina pectoris developed constant chest pain lasting one day and followed by congestive heart failure and shock The initial set of tracings showed prominent QS_3 and QS_{aV_F} in the presence of left ventricular hypertrophy suggestive but inconclusive evidence of posterior infarction The pronounced depression of the RS T segments in Leads V_4 to V_6 is consistent with left ventricular hypertrophy but possibly a reciprocal change associated with acute posterior infarction Note that the transitional zone was between V_3 and V_4 The second set of tracings shows a shift of this zone to a location between V_5 and V_6 mildly suggesting right ventricular dilatation and acute cor pulmonale The final set shows left bundle branch block with late intrinsicoid deflections in Lead V_6 Postmortem examination showed a large acute infarct of the posterior wall of the left ventricle and the posterior aspect of the septum, scattered fibrosis throughout the rest of the myocardium and a small infarct in the lower lobe of the right lung The QRS complex measured 0 10 second in the initial set, 0 12 in the second and third sets It is possible that if additional leads were recorded to the left of V_6 in the second set left bundle branch block would *have been established at that time*

assume acute infarction (Fig 154), especially if the degree of elevation recedes while the patient is under observation Here a series of tracings reveals definite and characteristic changes The bundle branch block in this case was also transient so that curves could be studied in block and out of block Both the early and the late abnormalities of muscle injury are apparent In the vast majority of cases of left bundle branch block and myocardial infarction, however, the electrocardiogram shows the fixed changes of left bundle branch block, and the decision regarding fresh myocardial infarction must rest upon other laboratory or clinical findings

Septal Infarction When the septum is infarcted the lesion is usually the septal component of a lesion of the anterior or posterior wall of the heart Those involving the posterobasal portion of the septum are more frequently associated with auriculoventricular block With involvement of either the anterior or posterior part of the septum, bundle branch block is

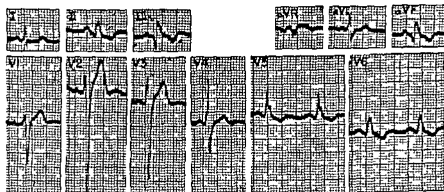


Fig 156 Left bundle branch block with old posterior myocardial infarction The QRS complex measures 0 12 second A late intrinsicoid deflection and a turret topped QRS complex in Lead V_6 suggests left bundle branch block The broad Q wave in Lead aV_F establishes the existence of an old posterior myocardial infarction The Q wave in Lead V_6 may be explained by septal infarction permitting passive transmission of right ventricular potentials to the left side of the precordium The patient was a 65 year old man who had an acute posterior occlusion five months previously Roentgen ray examination showed calcification in the descending branch of the left coronary artery

quite common (Figs 151 to 156) In rare cases the infarct may extend through the entire anteroposterior extent of the septum and involve the adjacent portions of the posterior and anterior walls (anteroposterior or Roesler-Dressler infarcts), producing changes in Lead aV_F and in some of the precordial leads Infarction of the septum is of particular importance in certain cases in explaining the presence of Q waves over a blocked ventricle in bundle branch block Here we are confronted with a complicated aspect of an already complicated situation In right bundle branch block the septum is activated, as normally, from its left to its right side If the septum is intact an initial upward deflection therefore appears in the right ventricular cavity and is transmitted to the right precordium If under the same circumstances the septum is infarcted, the left ventricular potentials, consisting of an initial downward deflection, may be transmitted passively

through the infarcted septum to the electrode over the right ventricle where Q waves are recorded (Figs 151 and 152) On the other hand, in left bundle branch block the septum is activated from its right to its left side If the septum is intact an initial upward deflection appears in the left ventricular cavity and is transmitted to the left precordium where an initial R wave is inscribed If, now, the septum is infarcted, the initial downward deflection of the right ventricular cavity may be transmitted passively through the infarct to the left ventricular cavity and thence to the left precordium where a Q wave may be recorded (Fig 156) A Q wave, therefore, is not expected over the right ventricle in right bundle branch block or over the left ventricle in left bundle branch block unless the septum is infarcted An appearance simulating left bundle branch block with septal infarction may be produced even if there is no bundle branch block and the septum is intact, by a subendocardial scar of the free wall of a markedly hypertrophied left ventricle or by extreme ventricular hypertrophy associated with hypertrophy of the septum (Fig 40)

Subendocardial Infarction Most transmural infarcts are wedge shaped with the broad part of the wedge on the endocardial aspect of the heart An electrode overlying the transmural part of an old infarct generally records a QS deflection, whereas one in relation to the epicardial aspect of the peripheral portion of this same infarct records a QR deflection, the Q wave being due to unopposed activation of the opposite ventricular wall, and the R to subsequent activation of the healthy subepicardial muscle overlying the infarct Changes similar to the latter have been recorded over scars which are purely subendocardial Acute subendocardial infarcts apparently can produce displacements of the RS T segment in either direction An extremely interesting subgroup in this latter category are those cases showing electrocardiographic changes similar to those classically developing during stress tests for coronary insufficiency, namely, depressed RS T segments over the precordium and an elevated RS T segment in Lead aV$_R$, without concurrent QRS changes (Fig 157) In some cases of this sort, developing characteristically under circumstances in which there seems to be a total rather than a segmental impairment of the coronary blood flow, postmortem examination may show rimlike subendocardial infarcts involving all or most of the subendocardial muscle The RS-T segment depressions in the precordial leads and the RS-T segment elevation in Lead aV$_R$ which faces the ventricular cavities, are probably due to the orientation of the injured area toward the endocardium The failure of Q waves to develop in these cases may be related to the fact that the subendocardium is extensively and uniformly infarcted, and that no part of the endocardium of the ventricular wall opposite the electrode is able to get a head start in being activated The subendocardial muscle itself or healthy islands of tissue interspersed with it must be capable of conducting the impulse to the healthy subepicardial muscle else the latter could not be activated Since QRS changes may not develop it is often impossible from a single tracing to decide if the changes represent subendocardial ischemia or infarction The persistence of these changes over a number of days,

especially if associated with correlative clinical or laboratory findings, would favor the diagnosis of infarction

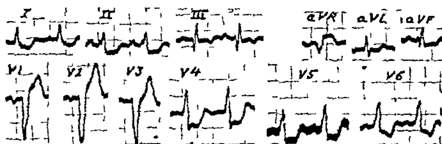


Fig 157 Acute subendocardial infarction The patient was a 53 year old man with a positive serologic test for syphilis and typical angina pectoris for ten months Following four days of extreme pain he was admitted in shock The tracings show depressed RS T segments in Leads I II aV_F and V_4 to V_6, and an elevated RS T segment in Lead aV_R The R waves are very small in Leads V_1 to V_3 Autopsy showed a fresh circumferential subendocardial infarct and syphilitic disease of the coronary artery orifices and right common carotid artery Tracings of this type may be recorded as a transient change during angina pectoris

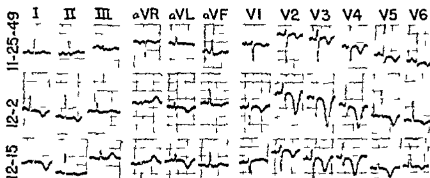


Fig 158 Myocardial infarction in the absence of QRS changes (ischemic T waves) The patient a 63 year old woman had repeated episodes of angina pectoris abating on rest or nitroglycerin during and shortly before her hospitalization The initial tracing (see also Fig 21) showed the characteristic changes of left ventricular hypertrophy but the T waves over the right ventricle were inverted rather than upright as one would expect them to be in left ventricular hypertrophy This change suggested anteroseptal myocardial ischemia or infarction Subsequent tracings showed waxing and waning of the inverted T waves and prolongation of the Q T interval (K = 0 49) suggesting but not proving anterior myocardial infarction without QRS changes This patient presented no laboratory evidence confirming infarction improved and was discharged The same changes in association with corroborative clinical and laboratory findings would be acceptable evidence for acute myocardial infarction Most cases with electrocardiographic changes of this type do well so that anatomic proof of infarction is generally lacking

Acute Myocardial Infarction Without QRS changes Attention has just been directed to the fact that subendocardial infarction may develop without significant QRS changes There appears similarly to be a small group

of cases of infarction resembling pericarditis in that RS-T and T wave changes develop without QRS changes, but differing from pericarditis in the degree of these changes In these cases a diagnosis of myocardial infarction may be justified if there are convincing clinical and laboratory findings sustaining that diagnosis In the absence of a clear cut clinical story and

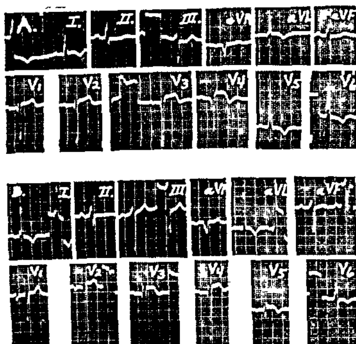


Fig 159 The fixed pattern of acute myocardial infarction in ventricular aneurysm The patient a 66 year old man had an acute myocardial infarction in 1946 The first set of tracings taken sixteen months later when the patient complained of dyspnea and angina show deep Q waves elevated RS T segments and terminally inverted T waves in Lead V_2 to V_4 As such these curves suggested acute myocardial infarction but there was no clinical or laboratory evidence suggesting this diagnosis Physical examination showed a systolic precordial bulge and fluoroscopy showed a paradoxical outward thrust with ventricular systole The second set of tracings was obtained eleven months later when the patient was in congestive failure They show essentially the same electrocardiographic features again in the absence of clinical or laboratory evidence for fresh infarction These are two of a long series of tracings showing the same appearance Right bundle branch block developed by March 1950 The patient died suddenly one month later Autopsy showed a large aneurysm of the left ventricle which was tremendously hypertrophied

particularly if fever, leukocytosis and elevated sedimentation rate are lacking, it is probably more accurate to refer to these changes as 'ischemic in nature (Fig 158) The prognosis appears to be good in either case Thus far there is a paucity of reports of autopsied cases which showed only RS-T segment and T wave changes

Ventricular Aneurysm RS-T segment displacements developing during

acute myocardial infarction are generally short-lived, subsiding within a few days or a fortnight In some cases, however, these changes persist indefinitely Since a Q wave is also present and the T wave may show late inversion (Fig 159), the appearance may be identical with that seen with fresh infarction Electrocardiograms taken on such individuals seen by a physician for some unrelated or related condition, especially if the latter

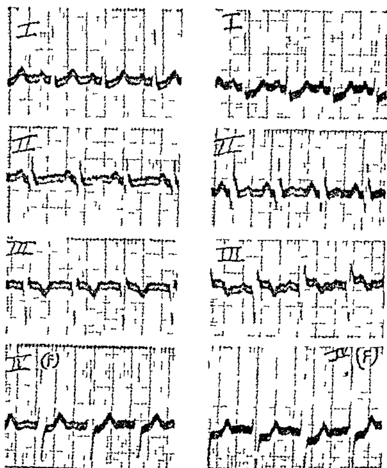


Fig 160 Exercise test for coronary insufficiency The patient was a young man 35 years of age who complained of pain in both arms only on effort The physical examination revealed no abnormality The first set of tracings was some what abnormal but not conclusive The second set taken five minutes after briskly walking up four flights of stairs shows conspicuous depression of RS T segments in Lead I and IV_F and elevation of RS T_3 This identified the symptoms as coronary in origin

resembles clinical acute myocardial infarction, can therefore mislead the physician into making the diagnosis of acute infarct However, under such circumstances, serial tracings will show an appearance which is quite fixed, whereas with acute damage one should find an unstable electrocardiogram Hence, although the combination of QRS, RS-T and T wave changes in a single tracing generally signifies acute infarct, strictly speaking, that diagnosis should be made only if sequential electrocardiographic changes are subsequently demonstrated. It has been shown empirically that this

finding of fixed QRS, RS-T and T wave changes is frequently associated with aneurysm formation in the myocardial scar as demonstrated by fluoroscopic, electrokymographic or pathologic examination and therefore may constitute a clue to the existence of that condition Why aneurysms should produce these changes is not clear It has been suggested that it may be due to the transmission to the electrode of the endocardial potentials produced in the hypertrophied ventricular wall opposite the aneurysm, to chronic coronary insufficiency or myocardial ischemia, or to a traumatic pericarditis The contribution of the muscle fibers remaining in the aneurysmal wall or of changes in the rim of intact muscle at the base of the aneurysmal sac is not known

"Stress Tests for Coronary Insufficiency

There are numerous instances in which the physician will be left in doubt as to whether a patient has angina pectoris or not In fact the differential diagnosis not infrequently may be serious coronary artery dis ease or no organic disease of the heart When the electrocardiogram is normal or equivocal under these circumstances, one is tempted to perform stress' tests to bring out significant alterations in the tracings For this purpose two methods are commonly used The first and simpler one is to take a series of electrocardiograms immediately after a brief effort or ' two-step test and to compare them with the control graphs (Fig 160) The second method is to take a series of tracings before and for five to fifteen minutes after allowing the patient to inhale 10 per cent oxygen In both instances characteristic anginal pain may be produced by these procedures From an electrocardiographic point of view the tests are regarded as positive evidence of coronary insufficiency if well marked deviation (usually depression) of the S-T segment results There are decided limitations to these tests as some cases of significant coronary artery disease will show a negative reaction and others with no coronary disease, especially patients who have been taking digitalis, may show sug gestive changes Furthermore these procedures, like all others in which one tries deliberately to reproduce anginal pain or coronary insufficiency carry some risk Rare fatalities or instances of coronary thrombosis with myocardial infarction have occurred Despite these possibilities such tests may be indicated on rare occasions

VENTRICULAR COMPLEXES IN ACUTE COR PULMONALE

Acute overloading of the right ventricle resulting from pulmonary embolism or other causes (e g massive collapse of several lobes of the lung) may produce electrocardiographic as well as clinical changes resembling those of acute myocardial infarction Myocardial infarction moreover, may be complicated by acute cor pulmonale and acute cor pulmonale may be complicated by coronary insufficiency In this difficult differential diagnosis the roentgen ray offers some help, particularly if oblique films are made of the region back of the heart However, it must be remembered that the roentgen ray diagnosis depends upon the demonstration of actual

infarction of pulmonary tissue, whereas the electrocardiographic changes probably depend rather upon the sudden increase in pressure which the right ventricle must support in some of these cases This may develop as the result of widespread spasm in collateral pulmonary artery branches even if only a small pulmonary artery branch is occluded and only an inconspicuous cone of pulmonary tissue is actually infarcted Thus the electrocardiogram and roentgen ray may supplement one another as diagnostic procedures However, pulmonary infarction or acute cor pulmonale may be present with a normal electrocardiogram or with normal roentgen ray findings

The changes which may be recorded are (1) *The development of a prominent* S_1 *and* Q_3 (Figs 161 to 163), which is probably related to rotation



Fig 161 Acute cor pulmonale (pulmonary embolism) The patient was a man 42 years old who was well except for an inguinal hernia His heart was normal Operation was performed on July 1, 1943 under spinal anesthesia He was doing well when on July 13 1943 while on a bed pan he had sudden pain in the left anterior chest dyspnea tachycardia and apprehension The pulse rate was 140 respiration 28, blood pressure 130 systolic and 80 diastolic His legs showed nothing abnormal X ray showed slightly cloudy left base and elevated left diaphragm On July 14 1943 bilateral ligation of the femoral veins was performed The patient ran a temperature of 101 to 102 F for five days and recovered The first tracings show changes indicative of acute cor pulmonale i e an S_1, a Q_3 a depressed S T_1, an elevated S-T_3 and an inverted T_3 Note the disappearance of these changes in the second tracing

of the heart on its own longitudinal axis in a clockwise direction so that right instead of left ventricular potentials are transmitted to the left shoulder The small R and deep S at Lead aV$_L$ are recorded unchanged in Lead I, thus deepening S_1 The small R and deep S at Lead aV$_L$ are recorded at Lead III as a small Q and tall R, thus tending to produce a Q_3 At the same time left ventricular potentials (qR) are transmitted to the left leg (aV$_F$) and are therefore recorded unchanged in Lead III This, therefore, contributes further to the development of a Q_3 (2) *Shift of the transitional zone to the left* may result from the chance variations in the heart s position resulting from its normal mobility However, conspicuous leftward migration of this zone toward the axilla (Figs 163, 164), especially if not otherwise explicable, is very suggestive of acute cor pul

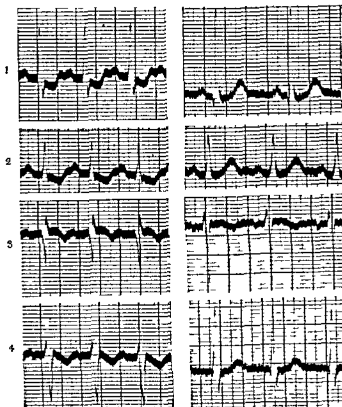


Fig 162 Acute cor pulmonale (pulmonary embolism) The patient was a woman 19 years old who had repeated pulmonary emboli post partum several days before the first tracing was made Note S_1 Q_3 depressed and inverted S T_1 and slightly elevated S T_3 These changes are not present in tracings made March 3 1943 (Courtesy of Dr Paul D White)

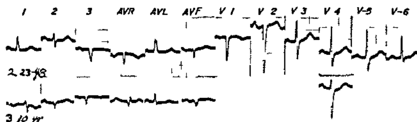


Fig 163 Acute cor pulmonale with leftward shift of transitional zone The patient was a 67 year old woman with carcinoma of the breast The initial pre operative electrocardiogram shows prominent QS_3 and QS V_F strong presumptive evidence of old posterior myocardial infarction The lower tracing taken two weeks postoperatively a few hours after the onset of shock and tachypnea shows the development of S_1 depression of RS T_1 and RS T_{aV_L} and a persistent QS in Leads III and aV_F The transitional zone which originally lay between V_2 and V_3 now was located to the left of V_4 The patient died eight hours later Postmortem examination showed emboli in both pulmonary arteries and an old posterior myocardial infarct.

monale This change is probably related to clockwise rotation of the heart on its longitudinal axis and posterior displacement of the apex of the heart, both probably brought about by ballooning out of the overburdened right ventricle, thereby displacing the septum to the left In

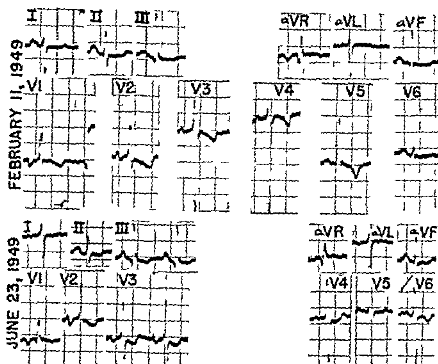


Fig 164 Displacement of transitional zone to left as clue to acute cor pulmonale The initial set of tracings (2/11/49) serve as control Leads V_1 and V_2 show tall R waves and inverted T waves indicative of right ventricular hypertrophy The S waves are larger than the R waves in Leads V_3 and V_4 Lead V_5 again shows larger R than S waves and an inverted T wave indicating that left ventricular potentials are now recorded The transitional zone then lies to the right of Lead V_5 The second set of tracings recorded when the patient was in acute failure with severe dyspnea and tachypnea again show deep S_1 and S_2 and inverted T waves over the precordium However since these changes were already present when the patient was compensated they cannot be adduced as evidence of acute cor pulmonale However at this time the transitional zone lay to the left of V_6 for left ventricular potentials are not recorded in the usual six precordial leads This was the only electrocardiographic clue to acute cor pulmonale The patient was a 26 year old man with rheumatic heart disease in congestive heart failure Postmortem examination showed that the heart weighed 780 gm Mitral stenosis and insufficiency aortic stenosis right and to a lesser extent left ventricular hypertrophy and multiple pulmonary emboli of varying age were detected

Figure 163 the preoperative electrocardiogram showed the transitional zone between Leads V_2 and V_3, the second tracing taken shortly after the onset of symptoms showed the transitional zone to the left of V_4 (3) *Inversion of the T waves over the right ventricle* (Fig 165) is probably related to a change in the repolarization process in the right ventricle (so-called

right ventricular strain), and may occur as an isolated finding without changes in the conventional or unipolar limb leads This change may help in the differential diagnosis from acute posterior myocardial infarction in which the T waves over the precordium are apt rather to be tall and upright (4) *Depression of the RS-T segments* in certain limb or chest leads (Figs 161 to 163) is probably related to subendocardial ischemia Since RS-T segments in Leads I and II may be depressed and in Lead III may be elevated, and Q3 may become prominent, the reason for confusion with acute posterior myocardial infarction becomes apparent It has been claimed that in acute cor pulmonale a prominent Q3 is not associated with a significant Q_{aV_F}, while this is theoretically sound, in actual experience it is not a thoroughly reliable differentiating point (5) *The*

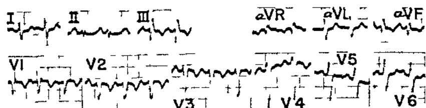


Fig 165 Inverted T waves over right ventricle in acute cor pulmonale The patient was a 74 year old man with increasing dyspnea for two months and an episode of substernal tightness lasting two hours one week previously admitted because of cyanosis dyspnea tachycardia (P 100–120) and tachypnea (R 30) Examination showed distant heart sounds pulsus paradoxus hepatomegaly very little orthopnea and clear lungs varicosities but no calf tenderness X ray showed an enlarged heart and several areas of fuzzy density in the right lung fields The electrocardiograms reproduced above show smartly inverted T waves in Leads V_1 to V_4 The S wave is prominent in Lead I There is a late intrinsicoid deflection in Lead V_1 Shortly after admission he suddenly went into shock with cyanosis blood pressure fell to 70 to 80 systolic he became much more cyanotic the neck veins became markedly distended and cardiac action ceased Postmortem examination showed massive pulmonary embolism obstructing both main stem arteries and bilateral popliteal and tibial vein thrombosis The heart weighed 580 gm and showed scattered myocardial fibrosis and coronary artery narrowing without occlusion In this case the inverted T waves and the tachycardia were the chief electrocardiographic clues to acute cor pulmonale

development of transient incomplete right bundle branch block (Fig 166) can also occur as a transient phenomenon in acute myocardial infarction In acute cor pulmonale it may be related to the increase in right ventricular pressure delaying the passage of the impulse along the right bundle branch

The changes that occur in acute cor pulmonale disappear when recovery takes place They may persist for days or weeks but often are present over only a very few hours, hence the need for frequent electrocardiograms during the few days following an acute episode suspected of being acute cor pulmonale In some cases several of the changes described may be recorded in combination, in a few cases a decision may have to be made upon the basis of a single change It should be emphasized that

any of the changes described as occurring in acute cor pulmonale may develop or be present in other conditions The T waves may be inverted over the right ventricle as a normal finding in hypokaliemia, in pericarditis, in myocardial ischemia and other conditions Incomplete right bundle branch block may be a normal variation which may occur with certain forms of congenital heart disease or during acute myocardial infarction The heart may rotate in a clockwise direction and point its apex posteriorly for other reasons than acute distention of the right ventricle In Figure 167, for example, are shown the tracings obtained from a woman of 82 whose heart occupied a most unusual electrical position in the chest She had a tremendous incisional abdominal hernia with a high

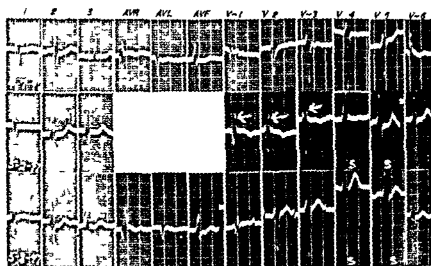


Fig 166 Incomplete right bundle branch block in acute cor pulmonale The patient was a 58 year old man in congestive heart failure with rheumatic heart disease involving the mitral and aortic valves The upper tracing shows right axis deviation and a late intrinsicoid deflection in Lead V_1 The second tracing taken during a spell of acute dyspnea, shows incomplete right bundle branch block Note RR complexes with late intrinsicoid deflections in Leads V_{1-3} The QRS complex measures 0 10 second in duration The lower tracing recorded six days later shows return toward the original form. Autopsy showed multiple recent and old pulmonary infarcts

left diaphragm displacing her heart upward It may be said, however, that the findings are of particular value if they have been observed to develop during the clinical episode under suspicion, and especially after abdominal operations or after traumatic injuries to the legs

Ventricular Complexes Resulting from Digitalis

In any consideration of the significance of an abnormal ventricular complex due regard must be paid to the possible influence of digitalis This drug produces two main effects It lengthens the P-R interval and depresses the R-T segment Figure 168 shows the gradual changes in the T wave resulting from a full dose of digitalis It will be noted that the T wave, particularly in Lead II gradually becomes depressed and finally

inverted The R-T segment attains a U shaped inversion with an upward concavity rather than with an upward convexity which characterizes the coronary form of the T wave Digitalis effects on the T wave are not always seen and do not appear with small doses They do not represent a toxic action of the drug as they appear when full therapeutic doses are given It requires two to three weeks for these effects to disappear after the drug is omitted

The changes described above indicate digitalis absorption and not digitalis poisoning The full fledged changes generally occur when the patient has been adequately digitalized, but it is wiser to depend upon clinical evidence to make the decision regarding adequate digitalization In general, the tracings are more helpful in telling whether the patient has received any digitalis at all Similar changes may be produced by other con

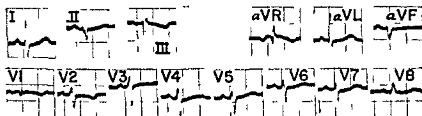


Fig 167 Normal heart in unusual electrical position The resemblance between Lead aV_L and Lead V_8 on the one hand and between Lead aV_F and Lead V_1 on the other indicates that the heart is in a horizontal electrical position The presence of S_1 and Q_3 indicate clockwise rotation (as viewed from the apex) of the heart on its longitudinal axis And the presence of deep S waves across the precordium and of a prominent R V_R show that the apex points posteriorly The displacement of the transitional zone to the left of Lead V_7 probably results from the last two factors Inverted T waves may be present over the right ventricle in women without heart disease All of the changes present may therefore be attributed to an unusual position of the heart The patient was an 82 year old woman with inoperable carcinoma of the pancreas who had a large incisional hernia of the left upper abdomen with marked abdominal distension Roentgen ray examination showed that the left leaf of the diaphragm was at the same level as the right Note that left ventricular potentials are not recorded until Lead V_8 is reached

ditions but in our experience, when this finding of digitalis T waves is at variance with the statement of the patient or his family, his physician much more often than not confirms the fact of recent digitalis medication

On the other hand, certain arrhythmias and conduction disturbances may indicate or suggest that the patient has received too much digitalis It is to be remembered at the outset that there is a drug factor and a myocardial factor in digitalis poisoning Certain damaged hearts seem to be particularly prone to develop evidence of overdosage with the drug, showing abnormalities on a dose which has no toxic effects in other hearts The characteristic change noted is some evidence of increased irritability of the ventricles These include ventricular premature beats, idioventricular rhythm (nodal tachycardia) or ventricular tachycardia Coupled ventricular beats (bigeminal rhythm), sustained or periodic, is

rhythm to a return to normal sinus rhythm until this has been proved electrocardiographically.

A rising ventricular rate may thus indicate the development of digitalis intoxication. It may also signal a worsening of the underlying disease process. It is sometimes exceedingly difficult to tell which. In such cases it is necessary to marshall all available clinical and laboratory evidence and make a decision, or, even at times, a gamble, one way or the other.

The ventricular tachycardia which develops as an evidence of digitalis overdosage *differs in no striking regard from that due to other causes.* One exception to this is the inception of a peculiar and rare type of ventricular tachycardia with alternating ventricular complexes, the so-called

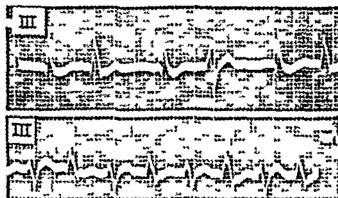


Fig. 170. Bidirectional paroxysmal ventricular tachycardia. The upper tracing shows auricular fibrillation with ventricular premature beats in bigeminal rhythm. The ventricular rate was 110. The lower tracing taken later the same day shows paroxysmal ventricular tachycardia with bidirectional complexes. The ventricular rate was 160. Note that the second and sixth (premature) complexes in the upper strip resemble the second, fourth, sixth and eighth complexes in the lower strip, while the fourth complex in the upper strip resembles the first, third, fifth and seventh complexes of the lower strip. In short, the two ectopic ventricular foci of the upper strip have usurped and share the ventricular rhythm in the lower tracing. This rhythm is pathognomonic *of digitalis overdosage.* The patient was a 56 year old man with rheumatic heart disease on digitoxin therapy for congestive heart failure. On discontinuing digitoxin for five days these toxic rhythms disappeared.

bidirectional ventricular tachycardia (Fig. 170). This rhythm is pathognomonic of digitalis intoxication.

PERICARDITIS

During acute pericarditis the electrocardiogram shows slight elevation *of* the RS-T segment. The leads in which this change is recorded vary with the area of dissemination of the pericarditis. In some cases these changes may be localized to one or two leads, e.g., Leads aV_L and I, in others they may be present in two or more limb leads and all or some of the precordial leads. Not uncommonly all three conventional leads show RS-T segment elevation *(Fig. 171A). If Lead aV_R faces the endocardial* aspect of an area whose epicardium shows RS-T segment eleva-

inverted The R-T segment attains a U shaped inversion with an upward concavity rather than with an upward convexity which characterizes the coronary form of the T wave Digitalis effects on the T wave are not always seen and do not appear with small doses They do not represent a toxic action of the drug as they appear when full therapeutic doses are given It requires two to three weeks for these effects to disappear after the drug is omitted

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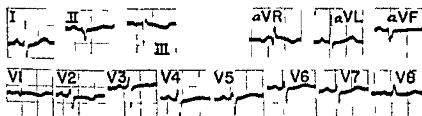


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very suggestive of digitalis overdosage but may develop in patients not receiving digitalis Multifocal ventricular premature beats generally indicates a marked degree of increased ventricular excitability This abnormal rhythm may be seen in grave myocardial disease, but much more often than not denotes digitalis poisoning

If the auricles are beating normally digitalis intoxication may be manifested by the various grades of auriculoventricular block With complete heart block the ventricular rate, though more rapid than the rate in complete heart block due to most other causes, is slower than the auricular rate The ventricles and auricles may beat independently of one another,

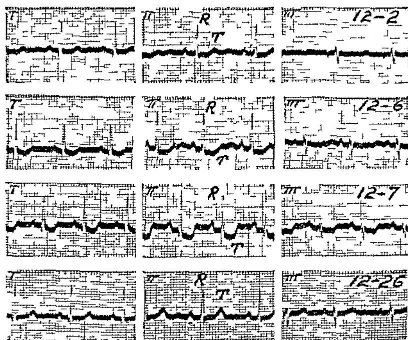


Fig 168 Digitalis effect One and one half gram of digitalis leaf was given between the first and third tracings Note the gradual inversion of the T waves in all leads and the return to normal in three weeks The RS T interval becomes convex downward (cupped depressed) unlike the upward convexity in coronary thrombosis (*Author's article in Oxford Loose Leaf Medicine* vol II)

not because of block between the auricles and ventricles, but for another reason, namely enhanced irritability of the ventricles In this case the ventricular pacemaker, be it in the auriculoventricular node or located lower in the specialized tissues of the ventricles or in ventricular muscle proper, takes on control of the ventricular rhythm because, although conduction between auricles and ventricles may still be intact, the auriculoventricular node or other ventricular pacemaker develops an enhanced automaticity and, so to speak, gets its ' punches in sooner ' than the impulses coming down from the auricles This is referred to as auriculoventricular dissociation It is characteristic of this condition that the ven-

tricular rate is more rapid than the auricular rate That the junctional tissues may still conduct the beat from auricles to ventricles may be demonstrated if there is a waxing in the automaticity of the auricular pacemaker Under such circumstances (Fig 169) a beat may slip through and be conducted to the ventricles, thus breaking up the ventricular rhythm This is referred to as interference dissociation Although this rhythm may be observed in other conditions it is an important clue to digitalis poisoning

When, on the other hand, the auricles are fibrillating digitalis poisoning may be manifested by the development of a slow regular rhythm, this represents complete heart block This slow regular rhythm (Fig 82) may



Fig 169 Interference-dissociation and reciprocal beating in digitoxin over dosage The first four QRS complexes are separated by intervals of 0 84 second Although preceded by P waves the latter occur at too short an interval to be con ducted The auricles and ventricles during that period are beating independently of one another the ventricles faster than the auricles This is auriculoventricular dissociation With the speeding up of the auricles the sixth auricular beat is con ducted through to the ventricles causing them to beat a little earlier This repeats itself in the next beat Thus the R R intervals between the fifth and sixth and between the sixth and seventh QRS complexes are 0 70 and 0 65 second respec tively This is interference The combination of these two mechanisms is inter ference dissociation Beginning with the second beat in the lower strip the ven tricular beats are conducted back to the auricles (retrograde beats note inverted P waves following QRS complexes) The fifth beat is conducted back to the auricles then some of the ventricular fibers having lost their refractoriness again back to the ventricles This is true reciprocal beating The sixth (re conducted) QRS complex is slightly aberrant The patient was a 58 year old man with hyper tensive cardiovascular renal disease who had received digitoxin in the dose of 0 4 mg daily for two or three months On withholding digitoxin normal sinus rhythm was restored

be detected on careful auscultation or electrocardiographic examination even if ventricular premature beats or coupled rhythm is present On the other hand as when coordinate auricular activity persists, the ventricles may become regular and rapid (idioventricular rhythm) The clue to the inception of either of these changes is the sudden regularization of a pre viously irregular heart It is true that normal sinus rhythm may be restored in patients with auricular fibrillation while receiving digitalis, apparently as a result of improvement in the circulation to the heart, but it should be remembered that the fundamental pharmacologic effect of digitalis is the perpetuation rather than the abolition of auricular fibrillation Hence one should in such cases hesitate to ascribe the development

rhythm to a return to normal sinus rhythm until this has been proved electrocardiographically

A rising ventricular rate may thus indicate the development of digitalis intoxication It may also signal a worsening of the underlying disease process It is sometimes exceedingly difficult to tell which In such cases it is necessary to marshall all available clinical and laboratory evidence and make a decision, or, even at times, a gamble, one way or the other

The ventricular tachycardia which develops as an evidence of digitalis overdosage differs in no striking regard from that due to other causes One exception to this is the inception of a peculiar and rare type of ventricular tachycardia with alternating ventricular complexes, the so-called

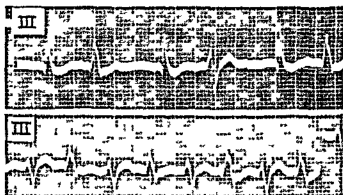


Fig 170 Bidirectional paroxysmal ventricular tachycardia The upper tracing shows auricular fibrillation with ventricular premature beats in bigeminal rhythm *The ventricular rate was 110 The lower tracing taken later the same day shows par* oxysmal ventricular tachycardia with bidirectional complexes The ventricular rate was 160 Note that the second and sixth (premature) complexes in the upper strip resemble the second fourth sixth and eighth complexes in the lower strip while the fourth complex in the upper strip resembles the first third fifth and seventh complexes of the lower strip In short the two ectopic ventricular foci of the upper strip have usurped and share the ventricular rhythm in the lower *tracing This rhythm is pathognomonic of digitalis overdosage The patient was* a 56 year old man with rheumatic heart disease on digitoxin therapy for con gestive heart failure On discontinuing digitoxin for five days these toxi rhythms disappeared

bidirectional ventricular tachycardia (Fig 170) This rhythm is pathognomonic of digitalis intoxication

PERICARDITIS

During acute pericarditis the electrocardiogram shows slight elevation of the RS-T segment The leads in which this change is recorded vary with the area of dissemination of the pericarditis In some cases these changes may be localized to one or two leads, e g , Leads aV_L and I, in others they may be present in two or more limb leads and all or some of the precordial leads Not uncommonly all three conventional leads show RS-T segment elevation (Fig 171*A*) If Lead aV_R faces the endocardial aspect of an area whose epicardium shows RS-T segment eleva-

tion, then RS T segment depression will be recorded there (Fig 171*B*) Although RS-T segment depression elsewhere is unusual and in fact may lead one to favor the diagnosis of myocardial infarction over that of pericarditis, any lead which happens to lie in relation to the endocardial

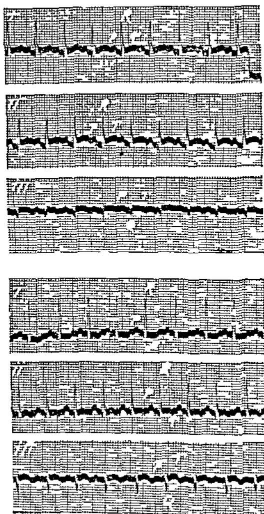


Fig 171*A* Acute rheumatic pericarditis Note the upward displacement of the RS T segments in all three conventional leads of the upper curves taken May 16 1935 These changes had disappeared on June 20 1935 when the second set was taken The patient had acute rheumatic pericarditis with effusion

aspect of a portion of the ventricular wall involved in the pericarditic reaction may show RS-T segment depression

Coincident with the RS-T segment elevation or during its subsidence a sharp, late inversion of the T wave develops The RS-T and T wave changes then have a contour identical with or closely resembling that frequently seen during acute myocardial infarction and not uncommonly

confused with it Attention to three points may help in the differentiation of these two conditions (1) In acute pericarditis a broad prominent Q wave does not develop since pericarditis does not lead to transmural myocardial damage and cavity potential is not transmitted to the epicardium A 'septal" Q wave may be present but this is due merely to the fact that the electrode faces the left side of a normally activated sep-

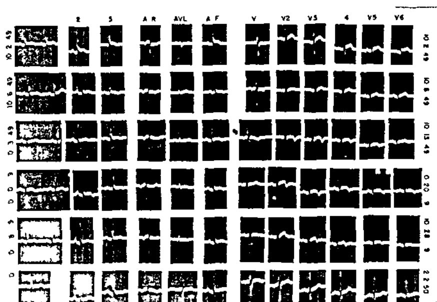


Fig 171*B* Acute benign pericarditis The initial set of tracings (10/2/49) shows elevation of the RS T segments in all conventional limb leads and in Leads V_2 to V_6 In the second set (10/6/49) the RS T segments have partially returned to the isoelectric line and the T waves are notched In the third set (10/13/49) the RS T segments are practically isoelectric and the T waves upwardly bowed and inverted resembling coronary T waves In the following set (10/20/49) the combination of minimal RS T and T wave changes is quite characteristic Clinical improvement coincided (10/28/49) with partial regression of these changes Follow up tracings three months later (2/2/50) show a normal electrocardiogram Note the failure of broad prominent Q waves to develop the slight degree of RS T segment elevation and the lack of reciprocal RS T segment changes to develop except in leads oriented toward the endocardium (Leads aV_R and V_1) The patient was a 25 year old man with sweats nausea malaise and pain in the back of the neck first suspected of having preparalytic poliomyelitis A pericardial friction rub Ewart s sign pulsus paradoxus and reflux distention of the cervical veins on hepatic compression were present He improved on penicillin and aureomycin therapy and was discharged well

tum The issue may theoretically be confused by the presence of a broad QS or QR wave residual from a previous infarct, but this situation is more a hypothetical than a real one The electrocardiographic diagnosis of acute pericarditis complicating acute myocardial infarction is little more than a guess, it may be suspected, however, from a temporary reversal or interruption in the characteristic sequence of electrocardiographic changes usually seen in myocardial infarction or from the presence of RS-T seg-

ment elevations at locations not showing QRS changes (2) The changes evolve somewhat more rapidly in pericarditis than in acute myocardial infarction, return of the RS-T segment to the isoelectric line generally occurring in the first two or three days (3) The RS-T segment displacements and T wave inversions are usually minor in degree Monophasic ventricular complexes or very tall or deeply inverted T waves have not been described in pericarditis

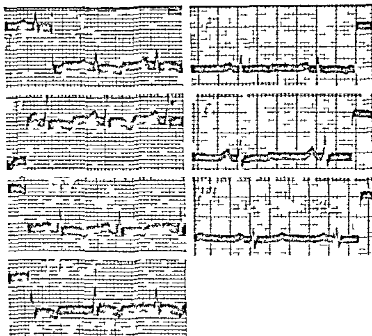


FIG 172 Constrictive pericarditis The patient was a woman 33 years of age who complained of fatigue dyspnea and swelling of the face Examination showed a quiet heart diminution of amplitude of ventricular contractions calcification of the pericardium edema of the face and increased venous pressure Resection of the pericardium was performed by Dr E C Cutler in February 1942 Recovery was satisfactory and several months later she was working full time The first set of tracings taken February 3 1942 show rather low QRS and inverted T waves in all leads The second set taken October 9 1942 show slight restoration of the T waves to a more normal form

A positive differentiation is not always possible Some cases of myocardial infarction, especially those rare cases in which the infarct is localized to a segment of the subepicardial layers, show only RS-T and T wave changes If the patient is first seen after the subsidence of the RS-T segment displacements it may be impossible to make a definite electrocardiographic diagnosis The changes must then be regarded as nonspecific, they might be consistent with myocardial changes due to many causes

T wave inversion may persist for weeks or, in rare cases as lo

or three months, but eventually the electrocardiogram resumes a normal appearance If the pericarditis is complicated by pericardial effusion the voltage of the QRS complexes may decrease, apparently, as in pleural effusions or ascites, because of local short circuiting

Ordinarily this sequence of events will not be observed in patients who show evidence of chronic adhesive or constrictive pericarditis The latter often show low voltage QRS complexes with or without T wave inversions (Fig 172)

It is generally considered that the electrocardiographic changes of pericarditis are due to acute subepicardial myocarditis Certainly the electrical changes recorded at the epicardium must have their origin in changes produced in or on the surface of heart muscle cells, and it must be very difficult if not impossible, either clinically or experimentally, to demonstrate restriction of injury to the pericardium and not involving the underlying myocardium Since the lesion may have progressed to a biochemical and not an anatomic stage, failure to demonstrate changes under the microscope would not exclude injury On the other hand, the fact that isolated injury of the epicardium is unlikely and that the ultimate origin of these electrical properties is in the myocardial cell does not disprove the theoretical possibility that 'pure pericardial injury might be associated with similar electrical changes

ELECTROLYTE IMBALANCE AND THE ELECTROCARDIOGRAM

Changes in the chemical environment of the heart muscle cell may also produce changes in the electrocardiogram Indeed, differences in electrolyte concentration between that on the inner and on the outer aspects of the semipermeable membrane constituting the surface of the cell account largely for the normal difference in potential across the normal resting cell We know little, except by inference, about the effect of variations in intracellular electrolyte composition, but we know a good deal more about the effect of changes in extracellular electrolytes upon the electrocardiogram

Hypocalcemia characteristically produces a prolongation of electrical systole manifested by a prolonged Q-T interval Mechanical systole may or may not show a concomitant lengthening This emphasizes the disparity between electrical and mechanical events and the danger of drawing mechanical implications from the electrocardiogram Prolongation of the Q-T interval is often observed in advanced renal disease with phosphate retention and reciprocal depression of the serum calcium level and in spontaneous or postoperative hypoparathyroidism (Fig 173) Prolongation of the Q T interval may also occur in alkalosis This change is probably referable to concomitant changes in extracellular ionized calcium, but its development in the hypochloremia of vomiting suggests a possible relationship to changes in intracellular potassium

The electrocardiogram may thus offer the first indication of lowering of the serum calcium level Although it has been claimed that the opposite chemical finding namely hypercalcemia, is manifested by a shortening of

the Q-T interval, recent experience suggests rather that prolongation of the Q-T interval, and of the QRS interval and depression of the RS-T segment may be associated with the high serum calcium levels in multiple myeloma

The average normal Q T interval can be determined from the following formula $K = \frac{Q\ T}{\sqrt{R\ R}}$ The normal figures for K are 0 39 for men and 0 44 for women It is generally unwise to report this as increased unless K exceeds 0 45 This figure is inaccurate at very slow and very rapid rates, in these instances the standards of Ashman should be consulted

The kidneys retain their ability to excrete potassium until relatively late, but in anuria from whatever cause, toxic accumulations of potassium may occur in the blood serum By virtue of the capacity of elevated serum potassium level to paralyze the heart this change may be a fatal complication of renal disease This development may be foreshadowed by taking

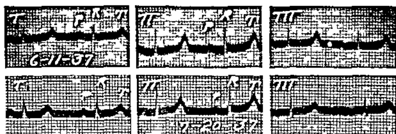


Fig 173 Prolonged Q T interval in hypoparathyroidism The patient was a man 68 years old with hypoparathyroidism Blood calcium on June 11 1937 was 4 7 mg per 100 cc and on July 20 1937 it was 9 3 mg per 100 cc The Q T interval despite very slight slowing of the heart decreased from 0 48 to 0 40 second The improvement occurred as a result of dihydrotachysterol

frequent electrocardiograms in anuric patients, thus providing the signal for timely and appropriate therapy The earliest change is the development of tall, pointed and narrow T waves (Fig 174) Later the Q-T interval, the QRS interval (Fig 39) and the P-R interval become prolonged At the same time the P waves become smaller, the R waves decrease the S waves increase in magnitude and the RS T segments become depressed Eventually the RS T segment forms an almost continuous line from the nadir of the S wave to the apex of the T wave and the P waves become unrecognizable (Fig 39) With further accumulations of potassium the rhythm may become grossly irregular and, on further disintegration of the ventricular complex the electrocardiogram may show the baseline to form a continuous sine wave Ectopic rhythms such as ventricular premature or escape beats and ventricular tachycardia may develop at this time The terminal mechanism in these cases may be ventricular standstill or ventricular fibrillation Although the intraventricular block developing in these cases is generally a diffuse affair affecting the ventricular myo-

cardium and specialized tissues alike, in a few cases bundle branch block has been recorded Some degree of elevation of the serum potassium level above the normal value of 4 to 5 milliequivalents per liter is generally necessary before any of these changes develop, but the parallelism between electrocardiographic appearance and the serum potassium level is only approximate Among other possible factors affecting this relationship is the serum sodium level An elevated serum sodium level antagonizes, while a depressed serum sodium level enhances these characteristic effects

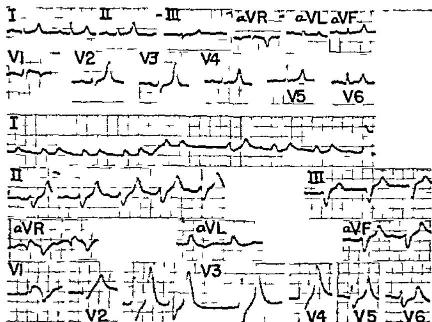


Fig 174 Potassium intoxication The patient was a 35 year old woman with anuria following premature separation of the placenta The first set of tracings showed first degree heart block and tall narrow pointed T waves the first clue to the existence of a high blood potassium level The second set of curves recorded five hours later shows much more pronounced changes including decreased height of the R waves increased depth of the S waves first degree heart block intraventricular block smooth slanting *RS T* segments and tall pointed *T* waves, most strikingly exhibited in the precordial leads Sinus arrhythmia and cardiac slowing are apparent in Lead I The serum potassium level at the time this second set of tracings was taken was 9 5 milliequivalents per liter Patient recovered following treatment by the artificial kidney and showed normal electrocardiograms at time of discharge

of potassium The effect upon potassium intoxication of variations in other ions such as calcium has not as yet been clearly demonstrated

Lowering of the serum potassium level, such as occurs in periodic paralysis, in certain cases of treated diabetic acidosis and in treated Addison s disease, is manifested by prolongation of the Q-T interval, by lowering, flattening or inversion of the T waves, by increase in the P R interval and by the development of U waves (Fig 175) The latter change may be a function of a *falling* serum potassium level for they have been

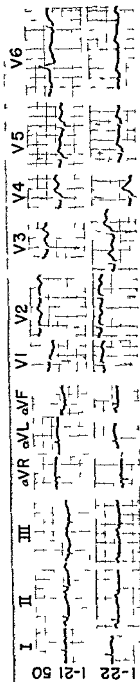


Fig 175 Hypokalemia The patient was a 67 year old spinster who had a sigmoid resection for carcinoma twenty eight years previously and a transabdominal total gastrectomy partial pancreatectomy and splenectomy on January 3 1950 Her post operative course was complicated by paralytic ileus and diarrhea and she presented a most difficult problem in the regulation of her electrolyte balance and maintenance of nutrition On January 19 her serum potassium level was 1 9 (normal 4-5 mEq/1) and chloride level 90 (normal 140 mEq/1), milliequivalents per liter On January 22 the corresponding values were 1 6 and 95 milliequivalents per liter respectively Note flattening or lowering of T waves depression of RS T segment prolongation of the Q T interval and the presence of U waves With subsequent return of serum potassium to normal levels the electro cardiograms became normal but due to somatic tremor are not satisfactory for reproduction

observed when the serum potassium was still elevated but falling It is to be emphasized that prolongation of the Q-T interval may be recorded with an elevated or depressed serum calcium level or with an elevated or depressed serum potassium level

WOLFF-PARKINSON-WHITE SYNDROME (ANOMALOUS ATRIOVENTRICULAR EXCITATION)

There is one peculiar type of electrocardiogram that occurs in a small group of individuals who, in about 50 per cent of the cases, are prone to have attacks of rapid heart action of one type or another (Figs 176 to 178) Although a few authorities have been willing to broaden the criteria upon which the diagnosis may be made, these patients generally have a shortened P-R interval measuring 0 10 second or less and a QRS complex broadened to 0 11 second or longer The mechanism of this disturbance is

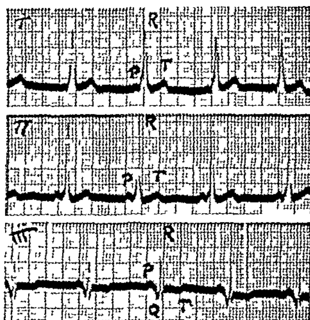


Fig 176 Wolff Parkinson White syndrome Note that the P R interval is very short (0 08 second) and the QRS complex is slurred and broadened (0 11 second) The broadening of the QRS complex is apparent only at the base producing a characteristic Eiffel Tower effect The initial or slow component of the QRS complex corresponds to activation of the ventricles over the hypothetical aberrant bundle Because the upper part of the QRS complex possesses a normal thinness the characteristic appearance may be missed in a cursory examination Peculiar curves of this type are seen in some individuals who have paroxysms of tachycardia but who are otherwise well

still debated but, following the lead of Scherf and Wolferth, it is suspected that it is due to conduction over an aberrant bundle of heart muscle such as the bundle of Kent, connecting auricles and ventricles and by-passing the normal auriculoventricular bundle Since this tract has no junctional tissue the impulse is not delayed In anomalous impulses activation of the ventricles is initiated over the aberrant route, probably in a direction from the epicardium toward the endocardium, but conduction down the normal pathway continues somewhat later Activation of the ventricles generally is completed over the normal route Thus the interval

from the beginning of the P wave to the end of the QRS complex is generally unchanged An adequate series of histologic studies in individuals with and without this mechanism is not as yet available to establish this anatomic mechanism It has been suggested that actual protoplasmic continuity of auricular and ventricular muscle need not be necessary, mere contiguity of auricular and ventricular muscle may be adequate to establish the mechanism A few cases have been reported in which patients



Fig 177 Wolff Parkinson White syndrome with anomalous complexes during paroxysmal rapid heart action The lower two sets of curves show typical short P R interval (0 10 second) and long QRS interval (0 12 second) The upper two sets of curves show a paroxysm of rapid heart action in which the individual ventricular complexes are similar to those recorded out of an attack The irregularity of the ventricles is probably due to auricular fibrillation During auricular fibrillation the impulses apparently invade the ventricles over the aberrant pathway These tracings simulate and were originally published as paroxysmal ventricular tachycardia The heart rate at first was 292 after 0 2 gm of quinidine it slowed to 231 and in less than one hour the rhythm was normal

without organic heart disease have died as a result of the tachycardia which may complicate this syndrome However, by and large these individuals carry on normally and have no organic heart disease Although most cases of this type have paroxysms of auricular tachycardia I have seen instances in which the attacks of rapid heart action were auricular flutter (Fig 178) or fibrillation (Fig 177) The type of ventricular complex seen in the paroxysm may be normal, in which case one might postulate conduction down the normal auriculoventricular node and

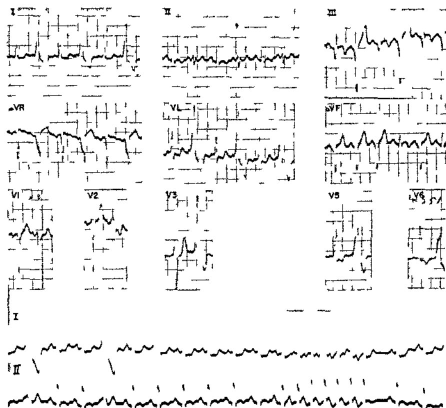


Fig 178 Wolff Parkinson White syndrome Anomalous atrioventricular excitation alternating with normal atrioventricular conduction Note that (with the exception of Lead aV_F) the first third and fifth complexes show normal P R interv ls and normal QRS complexes and are characteristic of left ventricular hypertrophy The second fourth and sixth complexes in these leads show a short P R interval and a lengthened QRS duration The slow initial portion of the QRS complex contributes to the characteristic Eiffel Tower effect If the anatomic explanation for this condition is accepted it would seem that for the most part every other beat is conducted earlier down the aberrant pathway than down the normal pathway whereas alternate beats start down the normal pathway earlier Note that the normal complexes in Lead aV_F do not show a broad or prominent Q wave This is evidence against the suggestion of old posterior myocardial infarction raised by the prominent Q and QS waves in the normal and abnormal complexes respectively in Lead III and by the broad QS waves of the abnormal complexes in Lead aV_F The direction of the QRS complexes is downward in Leads III aV_R aV_F V_1 and V_2 upward in Leads I aV_L and Leads V_3 to V_6 and equiphasic in Lead II This suggests that the general direction of abnormal excitation is from the right posterolateral to the left anterolateral aspect of the heart and toward the left shoulder at right angles to the Lead II line During the simultaneous recording of Leads I and II on a later occasion (lower sets) a short paroxysm of auricular flutter is recorded at a rate of 215 with 1 1 auriculoventricular response There is no important deviation of the spatial ventricular gradient between normal and anomalous complexes This accords with the hypothesis that the change from normal to anomalous complexes is merely one in the direction of invasion of the ventricles with no local changes in the responsiveness of the ventricles

bundle, or it may be anomalous, in which case the individual ventricular complexes resemble the aberrant complexes observed without tachycardia If one accepts the anatomic hypothesis, the latter type of paroxysm might indicate that the impulse enters the ventricle over the aberrant pathway It has been suggested that it is those patients who show both normal and anomalous complexes who are prone to have attacks of rapid heart action Quinidine, probably by virtue of its predominant effect in delaying conduction over the anomalous bundle, tends to favor conduction over the normal auriculoventricular node and bundle Exercise by its predominant effect in decreasing vagal tone and, frequently, atropine by paralyzing vagal nerve endings, enhance conduction over the auriculoventricular node and bundle Vagal stimulation by its predominant effect in slowing conduction in the auriculoventricular node favors earlier conduction down the aberrant bundle There are undoubtedly concealed instances of anomalous conduction which may be brought to light by various physiologic or pharmacologic procedures like those just enumerated There is a different group of individuals who show a short P-R interval (about 0 12 sec) with *normal* QRS duration also prone to paroxysmal rapid heart action It is important not to be misled into the diagnosis of anomalous atrioventricular excitation when the short P R interval-prolonged QRS complex is mimicked by a ventricular premature beat occurring only very slightly prematurely

The resemblance between the complexes of Wolff Parkinson-White syndrome and those characterizing myocardial infarction is only superficial It should be remembered that paroxysmal rapid heart action from whatever cause may be associated with chest pain It should not be concluded because rapid heart action and chest pain occur together that myocardial infarction is present When myocardial infarction complicates the Wolff Parkinson-White syndrome the electrocardiographic changes of infarction may be totally or partly lacking With anomalous conduction the impulse proceeds from the epicardium to the endocardium making the ventricular cavity initially positive thus a Q wave will be lacking at an electrode overlying a transmural infarct The situation is analogous to that in left bundle branch block in which the impulse travels from the right to the left side of the septum, similarly making the left ventricular cavity initially positive Since ventricular activation may be completed in a normal manner RS T and T wave changes may be detected in the anomalous complexes Be that as it may, examination of spontaneous or induced normal complexes may reveal decisive QRS, RS T and T wave changes of infarction

VENTRICULAR COMPLEXES IN OTHER CONDITIONS

There are numerous other conditions in which the ventricular complex alters its form In many the changes are not sufficiently characteristic or uniform to be diagnostic Further study will be necessary before such data can be standardized and before decisive conclusions can be drawn In the meantime it is important to be familiar with some of the peculiar-

ities that may arise Reference has already been made to the fact that inversion of T waves may be associated, among other causes, with ventricular hypertrophy or with electrolyte imbalance Frequently a definite cause for the inversion of the T waves cannot be established, yet one may be sure that they represent a pathologic state of the heart muscle In such cases it seems wise to refer to them as abnormal nonspecific change Markedly inverted T waves in all leads indicate a poor prognosis Some of these markedly abnormal ventricular complexes probably represent multiple areas of healed myocardial infarctions, some of which cases have been described pathologically as fibrous myocarditis

Flattening of the T wave and decrease in the height of the QRS complexes also occur in constrictive pericarditis (Fig 172), marked anemia,

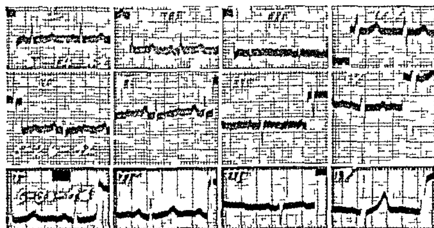


Fig 179 Abnormal form of ventricular complex (low voltage in beriberi heart) The patient was a man 33 years old who had been drinking heavily and eating very little He complained of weakness shortness of breath swelling of the legs and pains in the limbs He showed marked dilatation of the heart pulmonary congestion and some peripheral edema A control period of digitalis caused no improvement On a course of thiamine therapy the heart returned to normal size and all congestion disappeared The first set of tracings (before thiamine) shows very low QRS complexes which even in twelve days return to normal

beriberi heart (Fig 179), emphysema (180*A*), myxedema (Figs 131 and 180) and Addison s disease (Fig 180) In pulmonary emphysema low voltage in the limb leads may or may not be associated with low voltage in the precordial leads In pulmonary disease in general and emphysema in particular the T wave may be inverted in Lead I, but generally in these cases the T wave is upright in Leads V_5 and V_6 By contrast, inversion of T_1 resulting from anterolateral ischemia is generally associated with T wave inversion over the left ventricle Examples of low electromotive force in myxedema and in Addison s disease associated with myxedema are given in Figure 180 Although normal electrocardiograms may be recorded in Addison s disease, the frequent combination of low voltage, first degree heart block, inverted T waves and prolonged Q-T intervals

in this condition recalls the resemblance of these curves to those characteristic of hypokaliemia

Changes in the electrocardiograms are not rare during diphtheria Younger patients are more apt to show prolongation of the P-R interval

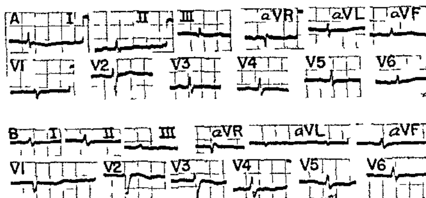


Fig 180 Low voltage electrocardiograms in endocrinopathies The upper set of tracings (*A*) was obtained from a 35 year old woman with severe myxedema Note that low voltage is recorded here in the chest as well as in the limb leads T_1 is inverted These changes are reversible on thyroid therapy The lower set of tracings (*B*) was obtained from a 24 year old man with Addison s disease associated with hypothyroidism Note the low voltage in limb and chest leads prolonged Q T interval and inverted T waves in the precordial leads These changes resemble those observed in hypokaliemia These changes may or may not be reversible on replacement therapy

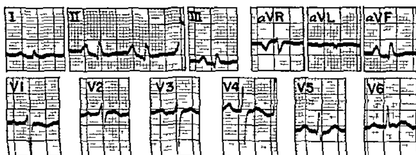


Fig 180*A* Chronic cor pulmonale associated with pulmonary emphysema and fibrosis The tracings show low voltage in the limb leads and prominent P waves in Leads II III and aV_F (P pulmonale) Note that while T_1 is low almost flat the T waves are upright over the left precordium this is evidence against ischemic anterolateral myocardial changes and more consonant with pulmonary disease The patient was a 59 year old man with long standing bronchial asthma bronchiectasis pulmonary emphysema and recent right sided heart failure The presence of low voltage in the limb leads suggests among other causes pulmonary emphysema

and more advanced stages of heart block, while the older more frequently show alterations in the RS T interval such as depression of the RS-T segment or inversion of the T wave

Inversion of the T wave in Lead I or Lead II is not normal and yet

its presence must not always be regarded too seriously When the inversion has the peculiar form described under myocardial infarction it may have important significance It should be remembered, however, that T waves of this type may be recorded in intracranial disease in the absence of heart disease Inversion of the T waves and prolongation of the Q-T interval resembling those associated with coronary artery disease may also be seen in left ventricular hypertrophy In general, as the R wave becomes taller and greater in area the T wave becomes smaller and inverted It is hoped that the study of the ventricular gradient in cases of this type might clarify the significance of these changes Moreover, the T waves can be diphasic or slightly inverted in neurocirculatory asthenia Cooling the apex of the heart by drinking cold water can produce inversion of the T wave Nitroglycerin or amyl nitrite can temporarily correct an inverted T wave in disease of the coronary arteries, and attacks of angina pectoris or a simple muscular effort may bring about inversion of the T waves in certain individuals It is obvious that abnormalities of the ventricular complex are frequent and require cautious interpretation

ELFCTROCARDIOGRAPHIC INTERPRETATION

Before leaving the subject of electrocardiography, it is necessary to express a word of caution against trying to read too much into the results of this examination Too often physicians attach significance to minor abnormalities It must be remembered that variations among normal hearts are very great Slight notching of QRS complexes, differences in amplitude of curves, left and right axis deviation and even flattened diphasic or slightly inverted T waves occur without organic heart disease Furthermore, when certain curves are diagnostic of myocardial disease they mav serve no further purpose in prognosis In a case of typical acute coronary thrombosis the curves may show very little abnormality or may be returning to a normal configuration and yet the patient may be doing poorly or may suddenly die It is generally idle to say that ' the electrocardiograms are getting better " It is more important to know what the patient is doing clinically than to know what further changes are going on in his tracings Although, in general, patients with inverted T waves in Lead I have a poor prognosis, there are a great many who live for years, and in fact there are some who have no clinical evidence of either angina or heart failure In the extensive use of electrocardiography there is a danger of prostituting the entire subject Attempts to interpret curves are being made when the leads were applied incorrectly or when the tracings were pasted upside down or wrong end to Artefacts are being interpreted as pathologic changes and many similar gross errors are being made Despite all this, with care and intelligence electrocardiography can be a most valuable aid in the care of patients with heart disease

PULSUS ALTERNANS

There is one disturbance in the mechanism of the heart beat that is truly not within the scope of electrocardiography because it is not diag-

nosed by this means However, it needs to be considered for it is important and can easily be recognized This condition is *pulsus alternans*, by which is meant an alternation in the strength of the beat when the rhythm is regular (Fig 181) In this condition the impulses of the heart arise regularly in the normal pacemaker and travel through the normal pathways The electrocardiogram is therefore normal Every other beat, however is stronger than the previous one It must be clearly differentiated from a pseudo-alternation due to prematurity of each second beat In bigeminy due to extrasystoles every second beat is also small but this results because the contractions occur slightly ahead of time and the ventricles contain less blood In true alternation the intervals between beats are equal and the weaker contractions are thought to be due to fatigue of the heart muscle or to impaired contractility It has been hypothesized that isolated bits of ventricular fibers here and there do not enter into systole because of fatigue so that each alternate systole is not so effective as the former one

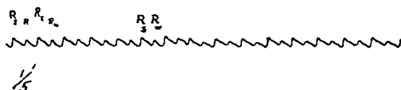


Fig 181 Pulsus alternans Tracing taken from the brachial artery which shows a regular pulse but waves alternating in strength R_S is larger than R_W (Author s article in Oxford Loose Leaf Medicine vol II)

Pulsus alternans can be recognized if a pulse tracing is taken from one of the peripheral arteries It can be felt at the radial pulse where every other beat will be weak Unless this sign is looked for or unless it is very marked it is generally overlooked Changes in the volume of the peripheral pulse produced by different phases of respiration often interfere with and obscure the regularity of the alternation At times the same alternation can be detected in the heart itself by noting an alternation in the intensity of the first heart sound or of an accompanying systolic murmur, or even in the force of the apex impulse In other words, the alternation may be seen heard or felt The simplest and most sensitive way of diagnosing pulsus alternans is with the sphygmomanometer While taking the blood pressure one should listen carefully and try to detect alternation of the sounds as the pressure falls and is just approaching the systolic level When the sounds first appear the pressure should be prevented from falling for several seconds If alternation is present the sounds will either alternate in intensity or only the stronger beat will be heard If the latter takes

place, then both sounds will become audible 5 or 10 mm lower but will still alternate in intensity This method is more sensitive and just as reliable as the pulse tracing in the diagnosis of pulsus alternans It should be sought for as a routine procedure in all patients with definite or suspected heart disease

In some patients pulsus alternans only appears for several seconds after a premature beat and then disappears Following the compensatory pause the first beat is always exaggerated but now the second beat is markedly diminished, the third is increased and so on for several cycles The extrasystole brings to the surface the tendency to alternation This has the same general significance as the more permanent form of alternation It also may appear during the very rapid rate of paroxysmal tachycardia in a patient without organic heart disease in whom no alternation *is present while the rate is slow (It then has no prognostic significance)*

Generally the electrocardiogram fails to show abnormalities that correspond with the pulse abnormalities Occasionally electrical alternation takes place, every other QRS complex being large and small Such alternation is supposed to have the same bearing on prognosis as that of the peripheral pulse Alternation of the P wave has also been noted during the rapid rate of auricular flutter

Pulsus alternans occurring in regularly beating hearts, apart from paroxysmal tachycardia, indicates heart muscle disease of a serious degree It is not seen in normal hearts It is most common in association with hypertensive heart disease and with disease of the coronary arteries but occasionally is found in valvular cases It is generally present when the rate is over 90 and may disappear as the rate falls to about 70 In fact, it is very rare when the heart is beating slowly Its importance lies in its prognostic significance The length of life after this sign is elicited is often not more than a year or two, although there are frequent exceptional patients who carry on quite satisfactorily for more than five years

PHONOCARDIOGRAPHY

Just as electrical instruments were devised to register the action currents in the heart, similarly apparatus is now available to photograph the heart sounds and murmurs These instruments are called *phonocardiographs* or *stethocardiographs* They are coming into use more and more, and it seems appropriate to touch upon the subject of phonocardiography rather briefly

Heart sounds and murmurs can be augmented in intensity many fold so that they can become audible in an amphitheater or they can be picked up by many listeners by means of appropriate electrical wiring and ear pieces In fact, heart sounds can be transmitted long distances These various procedures are used very rarely, but the future may see many developments little thought of at present What concerns us here is the photographic registration of the heart sounds as commonly obtained by the simple phonocardiographs in use

At the outset one might inquire as to what use phonocardiography

might have at present If the physician is hard of hearing the heart sounds can be intensified so that he may be able to detect sounds that he otherwise would not discover If hearing is normal it is doubtful whether any clinical interpretation of significance can be made from eliciting faint sounds with the phonocardiograph that are inaudible with the ordinary stethoscope There are instances, however, when even the expertly trained ear finds it difficult or even impossible to time accurately a sound or murmur in the cardiac cycle This is particularly true of the third heart

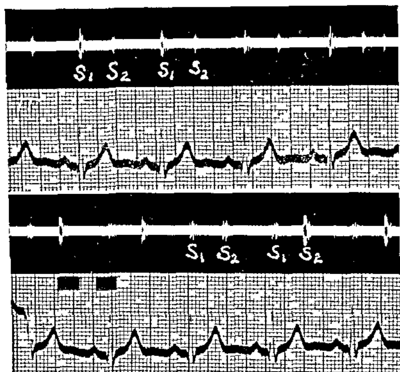


Fig 182 Normal heart sounds Simultaneous phonocardiograms and electrocardiograms of a normal man 29 years old The upper sound curves were taken from the apex and the lower from the pulmonary area Note that the first sound (S_1) is louder at the apex and fainter at the base than the second sound (S_2) The relative intensity of the two sounds varies both in normal and abnormal hearts

sound producing a gallop rhythm All physicians have occasionally called a murmur systolic in time erroneously instead of diastolic or vice versa The simultaneous registration of heart sounds and electrocardiograms obviates any such difficulties for it enables one to place any of the auscultatory phenomena in their exact portion of the cardiac cycle

The significance and composition of the heart sounds are being elaborately investigated by many students and will not be discussed in any detail here Suffice it to say that the first heart sound (lub) is made primarily by the closure of the mitral and tricuspid valves It is generally

thought that it is partly muscular in origin although some doubt this The second heart sound ("dub) is made by the closure of the aortic and pulmonary valves The interval between "lub" and "dub" is systole and that between "dub ' and ' lub is diastole In some normal hearts, especially in young persons in whom the rate is slow, a normal third heart sound may be heard in diastole The auricular contraction may produce a sound that is rarely audible though it may be present in phonocardiographic tracings When abnormal sounds or murmurs are heard it is obviously important to place them correctly in one part or another of the cardiac cycle

Figure 182 is an example of the normal heart sounds obtained in a regular normal heart It should be noted that the first heart sound comes directly after the QRS complex and the second sound at the very end of the T wave Figure 183 shows the character of the heart sounds in a grossly irregular heart (auricular fibrillation) The height of vibrations is

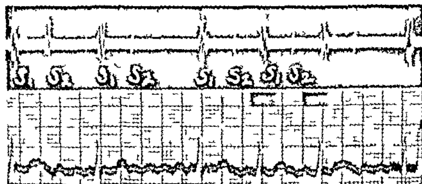


Fig 183 Irregular sounds of auricular fibrillation Note the gross irregularity of the rhythm Systole (S_1 to S_2) is fairly constant but diastole (S_2 to S_1) varies from cycle to cycle The patient was a woman 56 years of age with thyrotoxicosis

some indication of the loudness of the sounds Figure 184 is a clear example of a so called normal midsystolic gallop rhythm The extra or abnormal sound occurs in midsystole, i e, between the normal first and second heart sound This must be clearly differentiated from a diastolic gallop which almost always indicates a pathologic state, in which the third or abnormal sound occurs somewhere between the second and the following first heart sound (Fig 185)

Pulsus alternans, which has been discussed previously, is detected in the peripheral pulse Occasionally, however, the alternating quality of the strength of consecutive beats in a regular heart may be detected in the alternate intensity of the heart sounds This is well displayed in Figure 186 In the upper tracing the alternation in the heart sounds is accompanied by alternation in the form of the electrocardiogram There probably is 2 1 defective intraventricular conduction The lower tracing shows that the auscultatory alternation is still present when the electrocardiograms remain of the same form throughout This alternation in the loud-

ness of the heart sounds was first detected on routine physical examination Murmurs, if present, may alternate in intensity and the apex impulse may be seen or felt to alternate in strength

The character and intensity of the first heart sound is a matter of some importance in clinical auscultation We know it is accentuated in mitral

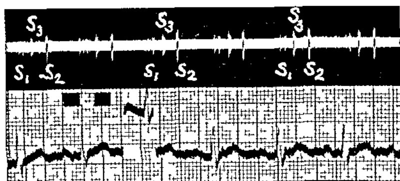


Fig 184 Normal midsystolic gallop Note that between the normal first heart sound (S_1) and the normal second sound (S_2) there is a definite third sound (S_3) which is even louder than the first sound The patient was a woman 35 years of age with rheumatoid arthritis but with no heart disease

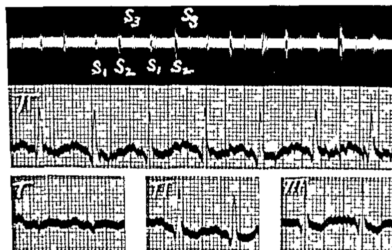


Fig 185 Diastolic gallop rhythm Note that the extra or third sound (S_3) occurs in diastole i e between the second and first sound This is almost invariably pathologic The patient was a man 60 years old who had severe congestive heart failure following an acute coronary thrombosis

stenosis, hyperthyroidism, and in some nervous and other states It may be diminished in intensity in emphysema pericardial effusion, in acute coronary thrombosis and, in fact, in some normal, healthy individuals Among the various factors that determine the intensity of the first heart sound, one that is most important is the P-R interval of that beat It has

been shown that when the auriculoventricular conduction time varies in different cycles (normally 0 14 to 0 2 second) the first heart sound will be loudest in those cycles with a P-R interval of about 0 04 to 0 08 second and that as the interval becomes longer the sound becomes more distant It follows that with intervals shorter than normal the sound is louder and with intervals longer than normal the sound becomes fainter than the normal first heart sound

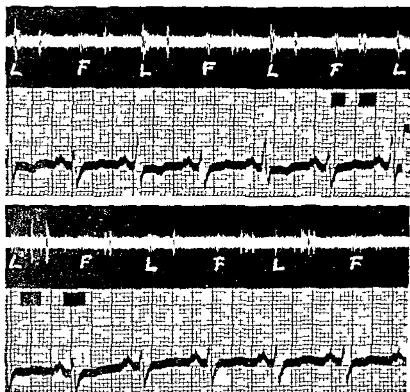


FIG 186 Ventricular alternation Note that alternate beats have faint (F) and loud (L) sounds In the upper strip the ventricular complexes also alternate but in the lower set the electrocardiograms are of constant type The alternation in the heart sounds was audible with the stethoscope and there was also pulsus alternans in the radial artery This man was 73 years old and had serious hyper tensive and coronary artery disease

This method, though not easily applicable, is in fact the only bedside method available in judging abnormalities in the relation between the time of auricular and ventricular contractions It may aid in the diagnosis of first, second and third degree heart block, nodal premature beats and paroxysmal ventricular tachycardia There is reason to believe that the changes in the intensity of the first heart sound in these conditions result because of differences in actual position of the auriculoventricular leaflets (mitral and tricuspid) at the moment the ventricles contract As the ven tricles fill in diastole the valve leaflets gradually float upward and then the auricles contract, pushing them deeper into the ventricular cavity, or

at least to a different position The exact position these leaflets will obtain will necessarily be different if the ventricles contract immediately after this alteration resulting from auricular contraction than if a longer interval intervenes between auricular and ventricular contractions In other words, it seems that the exact position of the mitral and tricuspid valves at the time of ventricular systole determines the loudness of the first heart sound The character of the snap will be different if it comes from a low position than from a high position This changing intensity of the first heart sound, while the ventricle is beating quite regularly, is the pathognomonic auscultatory sign of complete heart block and is clearly

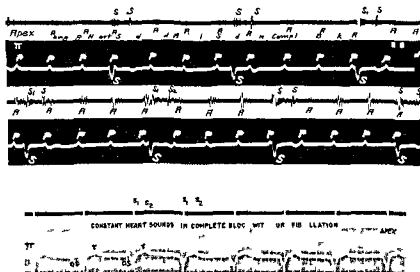


Fig 187 In upper tracing note changing intensity of heart sounds especially first sound (S_1) in different cycles Very loud sounds occurring with very short P R intervals When P and QRS occur simultaneously first sound is not increased Also note audible auricular sounds (A) Male age 75 years had Adams Stokes disease In lower tracing a case of complete block with auricular fibrillation shows no change in first sound The auricles are not contracting and thus there is no P R alteration

illustrated in Figure 187 Note that when the P waves come very close to the QRS the first heart sound is very loud and when the P-R interval is long the sound is diminished

Numerous other illustrations can be given showing the application of phonocardiography to the study of heart sounds Much of it is still in the speculative stage The above cases were examples in which the ordinary ear with a simple stethoscope was able to detect significant abnormalities and make correct diagnoses which were merely confirmed by the phonocardiograph Possibly much more valuable data will be obtained as such studies continue

Phonocardiography also affords a simple means of registering cardiac

murmurs With the present methods of amplifying sounds it is an easy matter to transform a faint murmur into a loud roar That does not help the practicing physician, for he will remain dependent for the most part on what he can detect with the ear and a simple stethoscope He will not be carrying around a complicated machine like the present stethocardiograph from house to house, though he may want to use it on occasions for special purposes or for investigative work Furthermore, the quality of the sounds is not quite the same when electrical apparatus is introduced Finally, most murmurs that will have any significance or of which any intelligent interpretation can be made will be audible with a stethoscope without electrical amplification If an extremely faint systolic murmur can only be detected with special apparatus it would have no clinical impor-

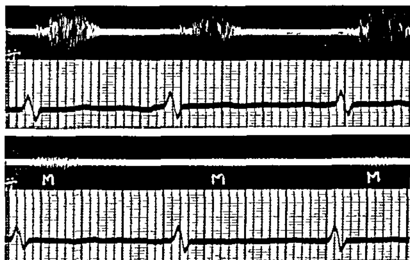


Fig 188 Loud systolic murmur of aortic stenosis transmitted to the elbow The upper tracing shows very loud systolic murmur (grade VI) in the aortic area The lower sound tracing was taken from the right olecranon process while the blood pressure cuff was inflated above the systolic pressure Note that the systolic murmur is still detectable (M) The patient was a woman 45 years old with aortic stenosis and angina pectoris

tance, because even slightly louder systolic murmurs (grade I or I minus) can be heard with the ear and yet often have no pathologic meaning However, we do know that a diastolic murmur that is diagnostic of mitral stenosis may become o faint that one observer may and another may not hear it, and, in fact, it may not be pre ent at all Under such circumstances amplification and registration of sounds may be valuable clinically Exploration of such problems is much in need at present

In a previous discussion (Chap 17) the significance of systolic murmurs was taken up At this point it seems appropriate to illustrate graphically one or two points concerning murmurs and to comment about their method of transmission I have long been convinced that there was something fallacious in the prevailing teaching concerning heart murmurs It

is generally thought that murmurs are transmitted *with* the blood stream A loud basal systolic murmur if heard in the carotid area is supposed to be indicative of aortic stenosis and an apical systolic murmur if trans mitted to the axilla indicative of mitral insufficiency These diagnoses have been made with greater assurance if such transmission were present than if not The point I should like to make is that transmission is dependent mainly, if not entirely, on the loudness of the murmur, and that transmission takes place from the point of maximal intensity (wherever the origin may be) in all directions Furthermore, there is evidence now available to show that transmission through bone is most likely of primary importance

Loud murmurs over the aortic area are often heard in the neck becau e they are near the neck and loud apical murmurs in the axilla because they are near the axilla There is no physical reason to explain the transmission of a murmur with the blood stream because the speed of transmission of sound is a great deal faster than the velocity of blood flow A murmur may be transmitted *in* but not *with* the blood stream It is not likely even that the transmission in the fluid media is important as will be seen by the following Figure 188 shows a loud systolic murmur, heard in the aortic area in a patient with marked aortic stenosis It was also present over the carotid arteries but was also readily heard at the right olecranon process, even when the blood pressure cuff was inflated to 220 mm of mercury (far above the systolic pressure of the patient) This means that the systolic murmur was transmitted through the bones of the arms for the blood supply to the elbow was entirely cut off

The fact that the loudness rather than its site of origin determines its transmission is shown by Figure 189 Here a very loud systolic murmur (grade VI) was heard all over the precordium, best in the third left interspace The patient had congenital interventricular septal defect This murmur was therefore made within the heart itself, and the current of blood producing the murmur flowed from left ventricle to right ventricle Despite this the murmur was heard in the carotid artery and also at the left elbow while the arterial supply was cut off This murmur also must have been transmitted through bone and not the blood stream Additional evidence that murmurs are well transmitted through bone is apparent in Figure 190 Here a loud musical aortic diastolic murmur was readily audible on top of the head and over the bones of the arms It is well shown in the phonocardiogram obtained from the right olecranon process

In general it may be stated that loud murmurs are transmitted in all directions from their maximum point of origin and that bone is probably the main pathway of peripheral conduction This explains why the systolic murmur of coarctation of the aorta is well heard in the interscapular region, for the site of formation of the murmur is deep in the chest and near the spine It follows that any murmur that is loud enough, no matter what its origin, may be audible over the carotid artery or even over the bones of the arms

Apart from the intensity of murmurs one derives very little diagnostic

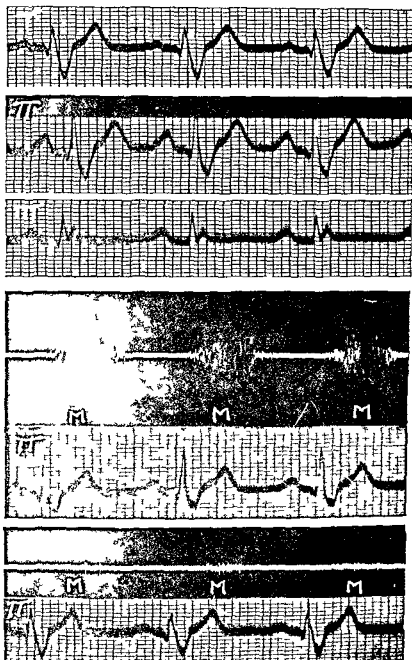


Fig 189 Loud systolic murmur of interventricular septal defect transmitted to elbow The upper three strips are the three electrocardiographic leads The middle set shows a loud systolic murmur from the third left sternal border The lowest set shows that the murmur (M) is audible at the left olecranon process with the blood pressure cuff inflated above the systolic pressure The patient was a man 21 years of age in good health showing definite evidence of congenital inter ventricular septal defect

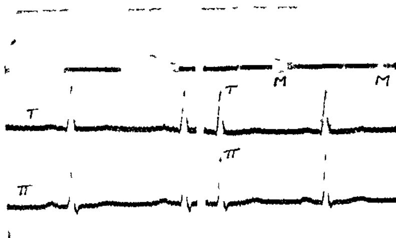


Fig 190 Loud aortic diastolic murmur transmitted to elbow The left set of curves shows loud musical aortic diastolic murmur taken simultaneously with Leads I and II The right set shows that the diastolic murmur (M) was audible over the right olecranon process with the arterial pressure occluded to that arm The patient was a man about 50 years old who had aortic insufficiency probably traumatic in origin

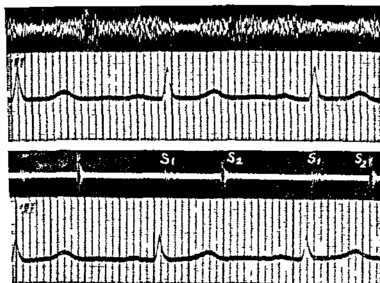


Fig 191 Continuous machinery murmur of patent ductus arteriosus The upper set shows a coarse continuous murmur increasing in intensity on approaching the second heart sound and again in the presystole obtained from the pulmonary area The lower tracings show normal first and second sounds (S_1 and S_2) without any murmurs following successful surgical division of the ductus The patient was a woman 24 years of age with congenital patent ductus botalli who is now cured

value from the quality of murmurs At times, however, the peculiar quality is of some interest This is particularly true of the continuous machinery murmur of patent ductus arteriosus This murmur often seems to envelop the heart sounds, becoming a little louder as the second heart sound approaches and continuing uninterruptedly after the second sound In this way it differs from the systolic and diastolic murmurs of aortic stenosis and insufficiency, in which there appears to be two separate components Figure 191 shows the disappearance of such a continuous murmur in a case of patent ductus arteriosus after a successful surgical division of the duct

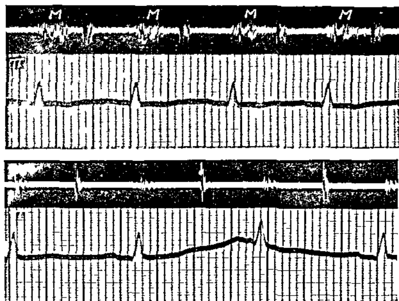


Fig 192 Systolic murmur produced by fever The upper set shows systolic murmur (M) in the pulmonary area while the patient s temperature was 104 F The lower set shows no murmur when the patient s temperature was 99 2 F This patient had general paresis but no heart disease and was receiving malaria therapy

One or two more simple observations may be appropriate at this time It has been mentioned in Chapter 17 that slight systolic murmurs may result from anemia fever, hyperthyroidism, physical exercise emotion and other noncardiac causes Figure 192 is a simple example of the production of a systolic murmur by fever This patient had no evidence of heart disease and showed no murmurs whatever She had general paresis and was receiving malaria therapy During the height of the fever (temperature 104 F) a distinct basal systolic murmur was present that entirely disappeared as the temperature returned to normal Finally, it is well to bear in mind that the disappearance of a murmur with inspiration does not necessarily mean that it has no clinical significance *Many faint* systolic murmurs will vary in intensity with respiration and some will dis

appear with a deep inspiration However, a faint diastolic murmur that is diagnostic of organic valvular disease may also disappear after a deep breath The procedure obviously decreases the intensity of some murmurs and the very faint ones may therefore become inaudible

Much more could be discussed concerning murmurs and their registration by phonocardiography but the entire subject is in the process of investigation and awaits further elucidation

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